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LUME 39 MARCH 1944 NUMBER 3

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TRAVENOUSLY INDUCED ANESTHESIA FOR OPERATIONS ON THE LARYNX UNDER DIRECT LARYNGOSCOPY

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ROCHESTER, MINN.

While many laryngologic operations can be rformed under indirect laryngoscopy, the ease th which suspension laryngoscopy can be emoyed at the present time makes indirect laryngscopy hardly necessary. Suspension laryngospy has certain advantages for both the patient d the surgeon. The patient finds it less trying an indirect laryngoscopy, particularly if he is rvous and excitable. The surgeon, in turn, not hampered during the operative procedure spasticity in the patient. This method is rticularly valuable when it is necessary to ke a specimen of the lesion and wait for a agnosis on the basis of a fresh, frozen section. the microscopic diagnosis makes treatment cessary, the whole procedure may be completed the one time while the patient is still asleep. lost intraoral laryngologic operations at the ayo Clinic are done employing the induction anesthesia to be described and using suspenon laryngoscopy.

The anesthesia for suspension laryngoscopy esents certain problems that are not encounred in anesthesia for operations elsewhere, for e following reasons. The reflexes in the laryngal region are particularly active. They disappear ly after deep surgical anesthesia has been acmplished, usually at the second or the third ane in the third stage, according to the Guedel assification. The muscles of the neck must be laxed thoroughly to permit a clear visual field. he vocal cords must be relaxed and open in der to expose a lesion so that it can be removed mpletely without undue trauma. While the ration of the operation on or about the larynx often only a few minutes, deep anesthesia ring the operative period is essential. The ality of the anesthesia therefore plays an imrtant part in producing satisfactory or unsatisctory operating conditions. Another factor of portance arises from the frequent use of the ctrocautery in this type of surgery, which eatly increases the danger of igniting explosive inflammable anesthetic mixtures.

In the past the induction of anesthesia by administration of ether was the method of choice and was perhaps the only method adaptable to this type of work. A brief résumé of its application should be of interest. The patient was anesthetized with nitrous oxide, oxygen and ether until deep surgical anesthesia had been accomplished, at which time a change to administration of ether by the drop method was made. The level of anesthesia was maintained by this method or deepened if necessary, during which time the gas machine was converted to deliver nitrous oxide, oxygen and ether directly by continuous flow. An intrapharyngeal nasal tube attached to a suitable connector for attachment to the delivery tube from the gas machine was then inserted through each nostril. The suspension apparatus was then inserted, and maintenance of anesthesia was accomplished by a continuous flow of nitrous oxide, oxygen and ether from the gas machine.

While the resultant anesthesia was usually satisfactory, the method involved certain drawbacks. Since the patient's mouth and the upper respiratory passages were open, it was difficult to maintain adequate alveolar concentration of the anesthetic mixture over periods longer than a few minutes. Consequently, with return of the laryngeal reflexes, anesthesia frequently became light and the operation had to be discontinued temporarily to permit further administration of drop ether. If the operation was long, this procedure often had to be repeated several times. When it was necessary to use the electrocautery, the ether bottle was removed from the gas machine, which was then flushed with nitrous oxide and oxygen. Under these circumstances the anesthesia rapidly became light, and if the cauterization was extensive, the patient often had to be reanesthetized. While few complications have been experienced from the use of the cautery in the path of the ether-laden exhalations of the patient, this practice cannot be said to be without hazard. In addition to these technical difficulties, the amount and the depth of the ether anesthesia were out of proportion to the duration and the extent of many of the operations. The postoperative recovery period

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was long, accompanied by prolonged periods of nausea and vomiting. Thus the induction and maintenance of anesthesia with ether left much to be desired.

For many years anesthesia induced and maintained by intravenous injection of the anesthetic agent was felt by many to be contraindicated for laryngoscopic procedures. The depth of anesthesia required to produce relaxation of the vocal cords was such that grave respiratory depression often occurred with occasional periods of apnea, hypoventilation and cyanosis. If a light plane of anesthesia was maintained, laryngeal relaxation often was unsatisfactory. The disadvantages of the method have been largely circumvented by topical application of an anesthetic agent to the glottis and by intrapharyngeal insufflation of oxygen throughout the course of the intravenously induced and maintained anesthesia.

The technic employed is as follows:

The patient is given preanesthetic medication with ⅙ to ¼ grain (0.011 to 0.016 Gm.) of morphine, 1/150 grain (0.00043 Gm.) of atropine and 1½ to 3 grains (0.1 to 0.2 Gm.) of pentobarbital sodium. Fifteen minutes before the operation is begun, the topical application of a 10 per cent solution of a cocaine salt is started. The patient is placed in the sitting position and is instructed to hold his tongue forward with a piece of gauze. The first step is to spray the pharynx lightly with the 10 per cent solution of a cocaine salt. A pause should then be made before further cocainization so that any untoward reaction to the cocaine may be observed. Hypersensitivity is rare. The pharynx and the region of the glottis and epiglottis are then thoroughly sprayed, following which the glottis is visualized by means of a laryngeal mirror. The cocainization is then continued by means of applicators. This includes applications in the pyramidal fossae, in the region of the glossoepiglottic fold and to the vocal cords. The applicator is held in each position for a few seconds. It is advanced a short distance through the vocal cords so that the greater portion of their mucous membrane is anesthetized. Usually two or three applications to the vocal cords are sufficient. However, the cocainization should be sufficiently thorough to interrupt reflex activity largely. If the cocainization is inadequate, it will be necessary to inject intravenously almost as much anesthetic as would be required if the cocainization had not been performed.

Instead of applicators a laryngeal syringe may be used to apply the cocaine solution. Not more than 3 cc. of a 10 per cent solution of a cocaine salt should be applied to the vocal cords by this method, preferably in divided doses. If the cords are open, care must be taken to prevent injecting the solution directly into the trachea.

The intravenous anesthetic agent is pentothal sodium in a 2.5 per cent solution in sterile distilled water. The preliminary dose of 4 to 6 cc. is administered, and when the patient is asleep and the muscles of the jaw are relaxed, an intrapharyngeal tube is passed by the nasal route. This is prepared by cutting a no. 6 or 6½ Magill intratracheal tube to the length of the distance between the ala of the patient's nose and the tragus of his ear. This tube when inserted extends a short distance into the oropharynx but does not obstruct the surgeon's field of vision. Through it oxygen is delivered to the patient during the entire course of the operation. Af placement of the tube, the suspension apparatus is i serted, and if masseteric or laryngeal relaxation is s inadequate, an additional 2 to 3 cc. of the solution pentothal sodium is administered. In some cases which the patient is resistant this dose may have to repeated once or twice with suitable pauses betwe Subsequent doses of 2 to 3 cc. are administered as quired during the operation. The average total d ranges between 1 and 1.5 Gm., and the total dose rare exceeds 2 Gm.

Intravenous anesthesia for operation in the lary differs somewhat from that for use in other regions the body. Some of the variations are worth stressi In the first place, the plane of the anesthesia must necessity be deeper than, for instance, in operations volving the skin and superficial structures. This res in a greater degree of respiratory depression than encountered in other operations. During operati involving the skin and underlying structures the an thesia may be lightened after the skin is incised, and during the course of the operation the anesthesia b comes light enough to permit muscular movement, t is not of great moment and interferes but little w the operation. In fact, the return of muscular tone often used as the criterion for the next injection. Su a scheme of administration will not give satisfact working conditions for the laryngeal surgeon, sin lightening anesthesia will result in spasticity of t vocal cords well in advance of the return of the reflex of the skeletal muscles. As a consequence, the degr of respiratory function must be the sole guide to t required depth of anesthesia and during the course the operation the respiratory function must be observ carefully. The lightening of anesthesia must be anti pated at all times by promptly noting an increase the amplitude of the respiratory movements and inje ing a further dose of 2 to 3 cc. at once. If this is don the vocal cords may be maintained well relaxed witho undue respiratory depression. If this scheme of admi istration is carried out, smooth satisfactory anesthes can be obtained. Since the intravenous anesthetizati for this type of operation is carried on at a general deeper plane than for operations on the skin, it is ex ceedingly important to keep a close check on t patient's pulse, color and respiratory function. It desirable to anticipate the completion of the operati procedure if possible in order to lighten the anesthes as soon as working conditions will permit.

COMMENT

This method of inducing anesthesia has bee a decided advance for many types of laryngea operations. It permits adequate relaxation i most cases; it obviates the hazard of fire an explosion and permits a more pleasant and com plication-free postoperative period for the patie than when ether anesthesia is used. To produc adequate relaxation without using excessiv doses of pentothal sodium, thorough cocainizatio of the glottic region is essential.

The successful and safe application of intra venous anesthesia for this type of surgical opera tion requires the services of an anesthetist skille in the induction of anesthesia by intravenou injection of pentothal sodium and in the manage ment of its effects.

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COCHLEAR MICROPHONICS

A CRITICAL REVIEW

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The electrical response from the cochlea has been used by many investigators to study the psychophysiology of hearing, and most of the theories in vogue at the present time are based on the results of experiments in which this technic was used. For this reason the "cochlear response" has a special significance for the otologist, as his final evaluation of the various theories rests on his evaluation of the experimental technic employed.

A developmental analysis of the phenomenon is herein presented. However, the conclusions arrived at are qualified by the reservation that while the ultimate psychologic discriminations must be imposed within certain limits by the events taking place in the middle ear, the cochlea and the auditory nerve, the presence of a particular discriminatory pattern at one level does not insure its presence at higher levels.

HISTORICAL REVIEW

In 1927 Forbes, Miller and O'Connor [1] reported an experiment in which they obtained electrical responses to acoustic stimuli in the decerebrate animal. Using a one stage electron tube amplifier and a string galvanometer, they recorded responses from the medulla oblongata and brain stem to acoustic stimuli of various types.

They obtained a fairly large and abrupt response to a sound of brief duration, such as a single click of a watchman's rattle. A weaker response but one showing oscillations correlated with the frequency of the stimulus up to 220 cycles per second was obtained to tuning forks and a card held in contact with a revolving cogwheel.

Three years later Wever and Bray [2] placed electrodes on the auditory nerve of a cat, amplifying the response to sounds, and found a correspondence between the frequency of the sound and the frequency of the response. They used a speaker in place of the string galvanometer of Forbes and co-workers, and words spoken into the cat's ear were reproduced with such clarity that it was possible to recognize the speaker by his voice.

Wever and Bray felt that they had ruled out the possibility of mechanical artefact by taking certain electrical precautions, and they attributed the response to action currents in the auditory nerve. They extended their study to other forms in order to establish this response as universal in organisms possessing auditory sense organs. Responses similar to those found in their original experiment with the cat were obtained from the guinea pig, the rabbit and the rat.[3] Likewise in the pigeon [4] they obtained responses from 100 to 10,000 cycles per second, although they found that the auditory mechanism of this bird is not as sensitive in regard to intensity as were the mammalian mechanisms studied. An electrical response to high frequencies could not be elicited from reptiles,[5] and crickets and katydids [6] gave only asynchronous nerve impulses.

These results indicate that wherever there is an acoustic mechanism the electrical phenomenon is also present and that the degree of development of this phenomenon is essentially correlated with the evolutionary level of the sense organ. The response from the mammals studied was such that the timbre of the voice could be recognized. Birds gave a similar response with the exception that sensitivity was poor; the relation between the magnitude of the cochlear response and the intensity of sound was found to be expressible as a power function with exponents

From Psychophysiological Laboratory, Occidental College, Los Angeles; Dr. Gilbert Brighouse, director.

1. Forbes, A.; Miller, R. H., and O'Connor, J.: Electrical Responses to Acoustic Stimuli in the Decerebrate Animal, Am. J. Physiol. **80**:363-380, 1927.

2. Wever, E. G., and Bray, C. W.: The Nature of the Acoustic Response: The Relation Between Sound Frequency and Frequency of Impulses in the Auditory Nerve, J. Exper. Psychol. **13**:373-387, 1930; Action Currents in the Auditory Nerve in Response to Acoustical Stimulation, Proc. Nat. Acad. Sc. **16**:344-350, 1930.

3. Wever, E. G.: Impulses from the Acoustic Nerve of the Guinea Pig, Rabbit and Rat, Am. J. Psychol. **43**:457-462, 1931.

4. Wever, E. G., and Bray, C. W.: Hearing in the Pigeon as Studied by the Electrical Responses of the Inner Ear, J. Comp. Psychol. **22**:353-363, 1936.

5. Wever, E. G., and Bray, C. W.: Auditory Nerve Responses in the Reptile, Acta oto-laryng. **16**: 154-159, 1931.

6. Wever, E. G., and Bray, C. W.: New Method for the Study of Hearing in Insects, J. Cell. & Comp. Physiol. **4**:79-93, 1933.

significantly below unity. It was found that reptiles respond only to low tones and that insects give only asynchronous responses. In the latter form the sensory apparatus is not a true auditory mechanism but consists of a tympanum and a set of sensory cells or scolophores situated on the foreleg of the insect.

The evidence obtained from the organisms studied is in accordance with comparative anatomic facts.[7]

ORIGIN OF COCHLEAR POTENTIALS

Many investigators thought, and some still do think, that the Wever and Bray results were due to electrical or mechanical artefacts. Kreezer and Darge,[8] for example, found that when they took precautions to avoid electrical and mechanical artefacts they could get only negative results, and they suggested electrical induction as the cause of the phenomenon.

To prove that the phenomenon has a biologic origin and that it is not due to physical artefacts, it is necessary to show exactly what that biologic origin is. Wever and Bray thought that the response was due to action potentials in the auditory tracts, but Adrian[9] suggested a microphonic action of the cochlea. Although later in collaboration with Bronk and Phillips[10] he reversed his view and ascribed the electrical response to the neural fibrils, his original hypothesis was taken up by others.

Bast and associates[11] placed chloroform in incisions made in the heads of cats so that the eighth and tenth cranial nerves lay in a pool of this substance. Instead of the expected diminution or obliteration of response, they found increased clarity in the reproduced sounds from the auditory pathways. They suggested that this was peculiar behavior for action potentials. It will be shown that in reality they were suppressing the action potential component of the Wever and Bray effect, leaving the electrical spread from the cochlea free from the distortion which that component introduces.

Guttman and Barrera[12] severed the eigh cranial nerve in cats subdurally and found th the electrical response still persisted eighte months after the operation. They attributed t failure of some investigators[13] to get an el trical response after severance of the eighth ner to poor surgical technic. This assumption is accordance with the facts, for the investigato who failed to get an electrical response did n go subdurally to sever the nerve, hence oth damage may have resulted, such as severance the internal auditory artery. It has been show by Davis and co-workers[14] that failure of t blood supply results in failure of the respons probably because of the death of the tissues i volved in its production.

Davis and Saul[15] were the first to take Adrian's hypothesis. They suggested that t response as observed by Wever and Bray had dual nature consisting of, first, action potenti and, second, a diffuse electrical spread from t cochlea. They originally based their theory o the following facts[16]:

1. Action potentials are sharply localized in t auditory tracts and follow the contralateral relati ship of these pathways—the cochlear potentials can picked up from any tissue of the head but are stronge when the petrous bone is included between t electrodes.

2. Action potentials disappear immediately at deat cochlear potentials persist for several minutes af heart action and respiration cease.

3. Frequencies above 1000 cycles per second are n reproduced by action potentials with accura Cochlear potentials reproduce with great fidelity to throughout the audible spectrum.

4. As stated above, cochlear potentials are stronge when the petrous bone is included between the electrod In the case of action potentials larger currents a obtained from the central tracts than from the eigh nerve. Adrian,[9] in suggesting an electrical spread fro

7. Hyman, L. H.: Comparative Vertebrate Anatomy, Chicago, University of Chicago Press, 1942, pp. 447-521.

8. Kreezer, G., and Darge, H.: Auditory Action Currents, Science **75**:105, 1932.

9. Adrian, E. D.: The Microphonic Action of the Cochlea: An Interpretation of Wever and Bray's Experiments, J. Physiol. **71**:28-30, 1935.

10. Adrian, E. D.; Bronk, D. W., and Philips, G.: The Nervous Origin of the Wever and Bray Effect, J. Physiol. **73**:2P-4P, 1931.

11. Bast, T. H.; Noir, R.; West, R.; Backu, O. L.; Krasno, M., and Eyster, J. A. E.: Electrical Currents Associated with Sound Reception by the Ear, Proc. Soc. Exper. Biol. & Med. **30**:638-640, 1933.

12. Guttman, J., and Barrera, S. E.: The Electric Potentials of the Cochlea and Auditory Nerve in Re tion to Hearing, Am. J. Physiol. **120**:666-670, 1937.

13. (*a*) Hallpike, C. S., and Rawdon-Smith, A. F The Wever and Bray Phenomenon: A Study of t Electrical Response in the Cochlea with Especi Reference to Its Origin, J. Physiol. **81**:395-408, 193 (*b*) Ashcroft, D. W.; Hallpike, C. S., and Rawdo Smith, A. F.: On the Changes in Histologic Structure and Electrical Response of the Cochlea the Cat Following Section of the VIII Nerve, Pro Roy. Soc., London, s.B **122**:186-197, 1937.

14. Davis, H.; Derbyshire, H. A.; Lurie, M. H., an Saul, L. J.: The Electrical Response of the Cochle Am. J. Physiol. **107**:311-332, 1934.

15. Saul, L. J., and Davis, H.: Action Currents the Central Nervous System: I. Action Currents of t Auditory Tracts, Arch. Neurol. & Psychiat. **28**:110 1116 (Nov.) 1932. Davis, H., and Saul, L. J.: Acti Currents in the Auditory Tracts of the Midbrain the Cat, Science **74**:205, 1931.

16. Davis, H., and Saul, L. J.: The Frequency Impulses in the Auditory Pathways, Am. J. Physi **101**:28, 1932.

region of the cochlea, maintained that the reason their presence in the neural elements of the auditory aratus was because the nerve acts merely as an rt conductor. The nerve, however, is the actual rce of action potentials, hence the larger the fibers the more fibers involved, the greater the potential—effect is additive.

Evidence Refuting the Neural Theory of Ori.—In order further to establish the electrical sponse from the cochlea as an entity separate om action potentials and to clarify the nature this phenomenon the following evidence is ed:

1. Polarity: Action currents always show gativity in the region of activity with respect an indifferent electrode situated elsewhere on e animal. On the other hand, the electrical gn of the first wave of the cochlear response pends on whether the first sound wave impinging on the ear drum is one of negative or one positive pressure. Negative pressure of the mpanum results in a positive cochlear potenl and vice versa.[17]

2. Vulnerability: Action potentials are rersibly deleted by narcotics and are suppressed, pecially in the higher auditory pathways beond the synapses of the cochlear nucleus, by ep surgical anesthesia.[16] Cochlear potentials, though depleted, persist for various periods of me after death; they can be abolished only by ilure of the blood supply or by pitting the basir membrane.[14]

3. Latency: Action potentials actually lag beind the cochlear response from 0.55 millisecond as much as 2.0 milliseconds, depending on the olarity and the intensity of the stimulus; this ct will be considered later in more detail when e function of cochlear potentials is determined.

4. All or None Law: The function of the imulus in neural physiology can be likened to man firing a gun: When the trigger is pulled, e gun fires with all the energy it has. The rce which the man exerts on the trigger bears relation to the resulting explosion once enough ressure has been applied to release the force ithin. In the case of a nerve fiber the potential enerated is not a function of the strength of the imulus but depends entirely on the nature and e immediate condition of the cell which genrates it. Once threshold is reached a nerve fiber acts in an all or none fashion. The number of bers participating in the response is a function the strength of the stimulus; hence action potntials are quantal in nature.

There is neither evidence of a steplike increase the magnitude of the cochlear microphonics nor a suggestion of an all or none relationship between stimulus and response. Furthermore, following a response a nerve fiber is inexcitable for a brief period; the cochlear response shows no such refractory period, a continuous response being elicited to prolonged stimulation.

5. Fatigue: Action potentials from the eighth nerve, as do those from all other medullated fibers, exhibit a characteristic which may be said to be a special form of fatigue. Davis and coworkers,[14] recognizing that the phenomenon was an adjustment to a new level of sustained activity and not an exhaustion of reserve material, gave it the name "equilibration." When the nerve is stimulated, there is a large initial volley of nerve impulses. Following this "on effect," the action potentials do not remain constant in size but shrink, first rapidly, then more slowly to a smaller amplitude.[17] This progressive diminution of response to an equilibrium level is considered to be due to the readjustment of the chemical dynamics of the nerve until a balance between the catabolic and anabolic processes is reinstated.

The cochlear response shows an "on effect" similar to that found in action potentials; in addition there is an "off effect." The origin of these characteristics does not lie in a chemical process as ascribed just now to action potentials but is due to an almost complete dampening of the basilar membrane. There is no evidence of fatigue or equilibration in the electrical response from the cochlea. Hughson and Witting[18] noted a depression of the cochlear response following supramaximal stimulation and termed it "auditory fatigue." A more descriptive term is "hysteresis" (as suggested by Stevens and Davis), because it emphasizes an analogy to physical processes and does not bear the connotation usually given sensory fatigue, that of temporary exhaustion of resources, but indicates that the process is akin to temporary pathologic damage. Supramaximal stimulation results in a depression of the response, and the threshold is found to be elevated—in other words, the sensory cells in question must return to their original state before the "normal," original curve of the response can be retraced. The process may be likened to an overloading of the system, and the period of recovery depends on the severity of the overload—from a few minutes to many hours.

On the contrary, the equilibration of action potentials is a function of frequency and not of intensity, there being a shrinkage with submaximal as well as with supramaximal stimulation. There is a fast equilibrium, complete in about

17. Derbyshire, A. J., and Davis, H.: Action otentials of the Auditory Nerve, Am. J. Physiol. **113**: 6-504, 1935.

18. Hughson, W., and Witting, E. G.: An Objective Study of Auditory Fatigue, Acta oto-laryng. **21**:457-486, 1935.

two seconds, dependent on the development of alternation of response in the fibers, and a slow equilibrium, complete in about seven minutes. Recovery requires about thirty seconds.[17]

Evidence For and Against the Hair Cell Theory of Origin.—Howe and Guild [19] in attempting to show that the Wever and Bray effect was not due to physical artefacts supplied excellent evidence supporting a non-neural theory of its origin and incidentally narrowed down the field of origin to the organ of Corti and its related structures. Using albinotic cats as subjects, they found that the audioelectric response to sound stimuli was absent. This type of animal is clinically known to be deaf, and examination reveals an extensive histopathologic abnormality of the inner ear involving practically complete absence of the organ of Corti with all its hair cells, marked pathologic changes in the macula sacculi and collapse of the cochlear duct and sacculus. However, the semicircular canals, the ampullae, the utriculus and the perilymphatic spaces are normal. A lack of response was never encountered where Corti's organ was demonstrable.

Lurie, Davis and Derbyshire [20] made observations of the electrical activity of the cochlea in certain pathologic conditions and came to the conclusion that the cochlear response depends on the organ of Corti because, first, it is absent where the organ of Corti is absent and, second, partial degeneration or deficiencies give partial and sometimes complete deficiency in the cochlear response.

Papers have been published in which the investigators reported recording the Wever and Bray effect either from other structures of the middle ear in the absence of the cochlea [21] or from the cochlea in the presence of complete destruction or degeneration of the hair cells of Corti's organ.[22]

I believe that these findings were either the result of mechanical or electrical artefacts due to failure to take precautions with apparatus or the result of poor histologic technic. These assumptions are based on evidence from more conclusive experiments, on certain inadequacies and in one case on misinterpretation of results. For example, the fact that Pumphrey [21] recorded microphonic potentials from lagena and perch, fish not possessing cochleas, is not sufficient evidence in that the sensory mechanisms of lower forms are not completely understood.

Eyster, Bast and Krasno [22b, c] attributed cochlear potentials to purely physical causes; they claimed that in a vibrating mechanism consisting of aqueous phases separated by membranes such as is the cochlea, electrical processes would invariably result from the physical pattern present. Yet their own paper refutes this hypothesis in that they found that drainage of the fluid contents of the cochlea (thus destroying the "physical pattern" they described) did not abolish the response. Furthermore, as they claimed that the hair cells were destroyed, and that Reissner's membrane, the eighth nerve and the cochlear duct are nonessential for the production of the cochlear response, no biologic apparatus capable of producing electrical potentials remains.

In the present study an attempt will be made to show not only that the organ of Corti is essential to the production of cochlear potentials but that these are produced by the hair cells of that organ.

1. Evidence from electrical experiments: Hallpike and Rawdon-Smith [13a] reported an experiment in which they made drill holes in the cochlear wall, filled the holes with drops of mercury and placed their electrodes in contact with the metal. They found that with an electrode near the apex the lower frequencies were transmitted with greater intensity than at the base and vice versa.

Culler [23] followed up this work, using guinea pigs, and obtained more specific results. The guinea pig's ear is particularly suited for experimental work in that the cochlea projects from the petrous bone and is not embedded in the bone as in the human ear. Culler exposed the organ and applied short lengths of sharp tungsten wire to twenty-five carefully localized points on the cochlear wall. Using an amplifier and a vacuum tube voltmeter, he measured the response of each point to each of twenty tones ranging from 125 to 7,000 cycles per second. He found that each point on the cochlear wall would respond maximally to a short range of frequencies. In other words, each frequency has its own focus of response within the coch-

19. Howe, H. A., and Guild, S. R.: Absence of the Organ of Corti and Its Possible Relation to Electric Auditory Nerve Response, Anat. Rec. (supp.) **55**:20-21, 1933. Howe, H. A.: The Relation of the Organ of Corti to Audio-Electric Phenomena in Deaf Albino Cats, Am. J. Physiol. **3**:187-191, 1935.

20. Lurie, M. H.; Davis, H., and Derbyshire, A. J.: The Electrical Activity of the Cochlea in Certain Pathologic Conditions, Ann. Otol., Rhin. & Laryng. **43**:321-343, 1934.

21. Pumphrey, R. J.: Microphonic Potentials from the Utricle, Nature, London **143**:898-899, 1939.

22. (*a*) Kobrak, H.; Lindsay, J. R.; Perlman, H. B., and Dubner, H.: The Effect of Limited Cochlear Lesions on Cochlear Potentials and Middle Ear Muscle Reflexes, Am. J. Physiol. **116**:93, 1936. (*b*) Eyster, J. A. E.; Bast, T. H., and Krasno, M. R.: The Origin of Cochlear Potentials, ibid. **119**:305, 1937; (*c*) Studies on the Electrical Response of the Cochlea, ibid. **113**:40, 1935.

23. Culler, E. A.: Symposium of Localization in the Cochlea, Ann. Otol., Rhin. & Laryng. **44**:807-813, 1935.

Furthermore, the localization of the frencies was such that the cochlea was graduated om high to low from base to apex of the cochlea spectively.

2. Evidence from surgical trauma: Using the chnic of drilling through the wall and damring the organ of Corti at various levels, Davis, urie and Stevens [24] recorded electrical potentials om electrodes placed on the round window and e neck of the animal to be studied. Electrical diograms were taken before and after the rgical operation. The exact location and exnt of each lesion was determined by postmortem stologic examination of serial sections of the chleas.

These investigators consistently found that, rst, high tones are localized near the basal end the cochlea, second, 2,000 cycles per second localized at the middle and, third, low tones e bunched quite closely toward the helicotrema. his is in substantial agreement with the evience from the electrical experiment described Culler.

However, surgical evidence alone is inconclue because of the complexity of the problems volved: Collapse of the scala tympani or of e scala vestibuli, or extensive hemorrhage into ese structures at the basal turn, or perforation the basilar membrane near the fenestra ovalis ould necessarily interfere with the physical ansmission of sound vibrations. Furthermore, o much resistance may be offered the flow of rrent if the round window is scarred or thickned, and short circuiting of the potentials genrated may result if there is an extensive lesion the basilar membrane.

The work of Davis, Lurie and Stevens is cited erely because it tends to confirm the evidence om other methods of investigation. It is not garded as conclusive in itself, because the comlicated nature of the surgical work involved renrs this type of evidence inadequate. The cochea is obviously not normal once it has been corched, drilled or otherwise injured. Considration of the laws of hydrodynamics reveals that e behavior of the liquid within a closed oscillaory system can be greatly modified by introducng even a minute hole anywhere in the containng walls. For these reasons, contrary evidence [25] will not be discussed in relation to the present problem.

3. Evidence from severance of the auditory nerve: Crowe [26] found that when the auditory nerve is cut at the internal auditory meatus, the cells of the spiral ganglion and the peripheral portions of the nerve fibers degenerate, which is contrary to Waller's law. Actually the sensory hair cells of Corti's organ ultimately completely degenerate and are absorbed as a result of traumatic change. Lurie [27] concluded that molecular disintegration of the hair cells takes place, the chemical constituents melting into the fluid of the scala media, producing toxic effects which cause further disintegration in other places, and so on. The traumatic change necessary to cause this degeneration is thought to be brought about by interferences with the blood supply caused when the internal auditory artery, which runs in close association with the nerve, is severed with it—a circumstance difficult to avoid.

Guttman and Barrera [12] found that whenever degeneration of Corti's organ is demonstrable, neither cochlear microphonics nor action potentials can be recorded. When degeneration of the nerve alone takes place, cochlear potentials are found but action potentials are absent. The fact that action potentials are never found in the absence of cochlear microphonics is taken as evidence that the electrical activity of the cochlea is essentially related to the processes involved in the excitation of the auditory nerve.

4. Evidence from stimulation deafness: Stimulation deafness may be of two types, first, that which occurs naturally as a result of profession [28] (e.g., that of boiler makers) and, second, that which is induced in the laboratory by the exposure of animals to loud protracted sounds.[29]

24. Davis, H.; Lurie, M. H., and Stevens, S. S.: s There Localization in the Cochlea for Low Tones? Discussion from Point of View of Animal Experimentation, Ann. Otol., Rhin. & Laryng. **44**:776-777, 935.

25. Hughson, W.; Thompson, E., and Witting, E. .: Is There Localization in the Cochlea for Low ones? Discussion from Point of View of Animal Experimentation, Ann. Otol., Rhin. & Laryng. **44**:777-92, 1935. Bast, T. H., and Eyster, J. A. E.: Is There Localization in the Cochlea for Low Tones? Discussion from Point of View of Animal Experimentation, ibid. **44**:792-803, 1935.

26. Crowe, S. J.: Anatomic Changes in the Labyrinth Secondary to Cerebellopontile and Brain Stem Tumors, Arch. Surg. **18**:982-991 (April) 1929.

27. Lurie, M. H.: The Degeneration and Absorption of the Organ of Corti in Animals, Ann. Otol., Rhin. & Laryng. **51**:712-717, 1942.

28. Wolff, D.: Microscopic Examination of Human Labyrinths from Patients Exposed to Loud Noises, Arch. Otolaryng. **36**:843-852 (Dec.) 1942.

29. Upton, M.: Functional Disturbances of Hearing in Guinea Pigs After Long Exposure to an Intense Tone, J. Gen. Psychol. **2**:397-412, 1929. Finch, G., and Culler, E.: Effects of Protracted Exposure to a Loud Tone, Science **80**:41-42, 1934. Horton, G. P.: The Effect of Intense and Prolonged Acoustical Stimulation on the Auditory Sensitivity of Guinea Pigs, J. Comp. Psychol. **18**:405-418, 1934. Wever, E. G.; Bray, C. W., and Horton, G. P.: The Problem of Stimulation Deafness as Studied by Auditory Nerve Technique, Science **80**: 18-19, 1934.

In both types degeneration of the hair cells of the organ of Corti has been demonstrated; no other histologic or anatomic abnormality is detectable. Kemp [30] proposed that it is the intensity-duration factor which causes the changes in the hair cells.

5. Evidence from congenitally deficient animals: Certain animals such as the albinotic cat and the waltzing guinea pig show varying degrees of deafness. Histologic study of the cochleas from these animals reveals partial or entire absence of Corti's organ.

As described before, Howe and Guild [19] could not detect a cochlear response in albinotic cats shown to be clinically deaf and showing, on microscopic study, complete absence of the organ of Corti. Davis, Derbyshire, Lurie and Saul [14] reported an animal which had one ear by which it could hear and which gave cochlear potentials and one ear in which neither function was demonstrable. The latter ear completely lacked Corti's organ.

Lurie, Davis and Derbyshire,[20] working with waltzing guinea pigs, found that cochlear potentials could not be elicited in animals showing complete degeneration of the hair cells of Corti's organ. In 1 animal showing degenerative changes only in the external hair cells throughout the organ of Corti, the threshold of response was raised.

6. Evidence from the application of certain chemicals: Fowler and Forbes [31] applied various chemicals (calcium chloride, quinine dihydrochloride and glycerin) to the round window membrane and recorded the effect on the cochlear response. They found an immediate loss for high tones progressing with time to the lower tones. The rapidity of progress and the degree of loss were proportional to the concentration used.

This progressive loss was interpreted as due to the effect on the end organs of an increased concentration of sodium or calcium traveling toward the apex of the cochlea and affecting the permeability of the cell membranes. Microscopic examination revealed, as was expected, progressive degeneration of the hair cells. The fact that the first demonstrable degeneration occurs in these cells supports the contention that the cochlear response originates in the hair cells of the organ of Corti.

30. Kemp, E. H.: A Critical Review of Experiments on the Problem of Stimulation Deafness, Psychol. Bull. **32**:325-342, 1935.

31. Fowler, E. P., Jr., and Forbes, T. W.: Depression of the Cochlear Response in Order of Frequency, Am. J. Physiol. **116**:51-52, 1936.

THE PROBABLE MECHANISM OF PRODUCTION

Having attributed the origin of the cochlear r sponse to the hair cells of the organ of Corti, becomes necessary to investigate the mechani involved. The question arises: Do the chara teristics of this phenomenon fit any known phys cal process?

Davis [32] suggested that mechanical vibrati causes the sensory cells of the organ of Corti develop an electrical potential between their u per and lower ends; in other words, the hair ce do not release stored energy but act merely as a electromechanical transducer converting mecha ical into electrical energy. The type of physic process which most satisfactorily fits the fac is the piezoelectric effect.

It has been found [33] that when pressure applied to certain substances having a particula type of crystal lattice, a dipole moment is ge erated. This is due to a separation of the ce ters of gravity of the position of the positive an negative charges in each molecule. The potenti created is essentially proportional to the distor ing force, the dipole moment being a product the multiplication of the value of the charges b their separation.[34]

Piezoelectricity is not confined to crystalli structures but may be produced in certain bio logic cells as a result of external stimulation. A with crystals, the magnitude of the distortion po tential is a function of the amount of mechanic distortion produced.

If cochlear potentials are generated by th piezoelectric effect, it is necessary that the mag nitude of the response conform to the law o the conservation of energy. On examination thi is found to be the case: No measurements hav yet been made of the electrical resistance betwee the two sides of the basilar membrane, but fro the resistance encountered in cellular structure elsewhere in the body it may be assumed to b about 1,000 ohms. Therefore, using the formul $P = E^2/R$ and substituting the value, 1,000 ohm for R and measuring the voltage, E, of the re sponse at a fixed frequency and intensity, it i found that the electrical energy elicited is neve greater than the power delivered to the ear.

In piezoelectric crystals the polarity of th charge depends on the sense of the mechanic

32. Davis, H.: The Electrical Phenomena of th Cochlea and the Auditory Nerve, J. Acoust. Soc America **6**:205-215, 1935.

33. Harnwell, C. P.: Principles of Electricity an Electromagnetism, New York, McGraw-Hill Book Com pany, Inc., 1938, p. 84.

34. Mason, W. P.: Quartz Crystal Application Bell Syst. Tech. J. **42**:178-188, 1943.

ess. It will be remembered that the electrical
n of the first wave of the cochlear response
ends on whether the first sound wave imping-
on the ear drum is one of negative or one of
itive pressure—negative pressure resulting in
ositive cochlear potential and vice versa. As
round window becomes negative, the oval
ndow becomes positive,[17] suggesting that the
sides of the basilar membrane develop op-
ite electrical charges in response to mechan-
pressure on the tympanum. This is taken
supporting evidence that the dynamism of
hlear potentials is the piezoelectric effect.
This hypothesis is not regarded as conclusive
is presented because it seems to fit the evi-
nce best.

THE FUNCTION OF COCHLEAR POTENTIALS IN AUDITION

For some time it was thought [35] that cochlear
entials represented the mechanism of stimula-
n of the terminations of the auditory nerve.
cent evidence renders this assumption highly
probable. For example, it was shown by An-
eev, Gershuni and Volokhov [36] that when the
ditory nerve endings were stimulated by alter-
ing currents in human subjects whose coch-
s were destroyed, all frequencies sounded es-
ntially alike; the sensation was one of noise.
is not only suggests that cochlear potentials
not stimulate the auditory nerve but indicates
t the discrimination of pitch is a peripheral
her than a central process.

The latency between the cochlear response and
e action potentials is of the order of 1 milli-
cond, a period far too long for electrical con-
ction time, but if the electrical potentials do
t stimulate the eighth nerve, it is difficult to
e just what physiologic function they perform.
believe that it is not essential that they have
y functional significance; the piezoelectric ef-
ct may be an epiphenomenon, an accompani-
ent of mechanical distortion of the symmetry in
lls of semisolid nature happening to contain
ganized oriented molecules.

THE ELECTROPHONIC EFFECT

A characteristic of the piezoelectric effect is its
versibility. Thus, if a quartz crystal is placed
an electric field, the crystal either expands or
ntracts, depending on the polarity of the elec-
tric field. If the theory that the ear acts as an electromechanical transducer of the piezoelectric type is correct, the converse of the effect should be observed—electrical stimulation of the cochlea should result in mechanical distortion of the sensory cells.

Volta [37] as long ago as 1800 recorded that on placing charged metal rods in his ears he received, in addition to a jolt, a bubbling sound in his head. It has since been found that when the normal ear is stimulated by alternating currents all frequencies within the audible spectrum are quite distinctly perceived. Gershuni and Volokhov [38] placed an electrode in an isotonic solution of sodium chloride poured into the external auditory meatus, and the other electrode elsewhere on the body. They found that up to the intensity at which their subjects received a shock all frequencies above 100 cycles per second were distinguishable.

By making certain checks they ruled out the possibility that the effect was due to oscillations arising in the electrode inserted in the liquid or to vibration of the components of the middle ear. They suggested that the phenomenon was a result of mechanical vibration in the cochlea due to the presence of an electric field.

Stevens [39] repeated the experiment and found that tones heard by electrical stimulation lack the purity of tones heard in the usual way, there being enough distortion to introduce strong harmonics when the ear is stimulated by a purely sinusoidal current. He suggested that this effect was a result of electrical rectification—that is, the current passes the boundary between various types of surfaces more easily in one direction than in another. He based this assumption on the fact that counter rectification reduces the harmonics heard.

If the distortion element can be disposed of in this manner, and the evidence is strongly in favor of such a hypothesis, the electrophonic effect can be taken as a cogent argument in support of the theory which attributes the generation of cochlear potentials to the piezoelectric process.

THE COCHLEAR RESPONSE AS AN INDEX OF HEARING

The cochlear response has been used by many experimenters to investigate both the quantitative and the qualitative aspects of hearing; how-

35. Hallpike, C. S., and Hartridge, H.: Electrical
imulation of the Human Cochlea, Nature, London
0:192, 1937.

36. Andreev, A. M.; Gershuni, G. V., and Volokhov,
A.: On the Electrical Excitability of the Human
r: On the Effect of Alternating Currents on the
fected Auditory Apparatus, J. Physiol., U. S. S. R.
:250-265, 1935.

37. Volta, A.: On the Electricity Excited by Mere Contact of Conducting Substances of Different Kinds, Phil. Tr. Roy. Soc., London **90**:403-431, 1800.

38. Gershuni, G. V., and Volokhov, A. A.: On the Electrical Excitability of the Auditory Organ on the Effect of Alternating Currents in the Normal Auditory Apparatus, J. Exper. Psychol. **19**:370-382, 1936.

39. Stevens, S. S.: On Hearing by Electrical Stimulation, J. Acoust. Soc. America **8**:191-195, 1937.

ever, the method has been criticized by some workers who feel that the response does not necessarily reflect all the psychophysiologic mechanisms involved in audition. Admittedly, little is known of the processes in the higher centers because of the experimental difficulties involved, but the cochlear response appears to reflect the function of the mechanisms which are known to be peripheral.

The objection that the cochlear response cannot be considered an index of hearing because it appears to have no physiologic function may be answered by an analogy: The movements of a runner are best analyzed by watching the man himself; however, should this be impossible, his shadow, if it were not distorted, would serve as a reasonable substitute.

As far as the qualitative aspect is concerned, aural microphonics reproduce with a fair degree of fidelity the quality of speech or music, once the distorting influence of the ever present action potential component is suppressed by anesthesia or cooling of the eighth nerve. The quantitative aspect is more complex.

The voltage of the cochlear microphonic increases as a continuous function of the intensity of the stimulating sound, and at low intensity, provided certain interfering factors are eliminated, the potential is directly proportional to the amplitude of the stimulus. However, as the intensity is increased, the curve relating stimulus to response becomes bent toward the intensity axis. This deviation may be a result of two factors: First, the phase relation between cochlear and action potentials may produce an interference pattern which either reenforces or decreases the response, and this phase relation is directly related to the number of fibers stimulated, which in turn is a function of intensity; second, the muscles of the middle ear contract reflexly in response to sounds, and the strength of the reflex is proportional to the intensity of stimulation.[40]

Beyond a certain intensity of stimulation, depending on the frequency, no greater electrical response can be elicited, the maximal potential being developed at about 100 decibels above the threshold of the microphonic at 1,000 cycles per second.[41] This is a characteristic of piezoelectric materials; once a certain degree of mechanical distortion is reached, increase in torsion fails to increase the potential generated. In view of these facts it is suggested that when the hair cells of Corti's organ reach a maximum of distortion, increase in stimulation fails to induce a greater electrical response.

The cochlear response, even if epiphenomenal, is regarded in the present study as furnishing a reliable index to the peripheral processes involved in hearing with the qualification that when some satisfactory method is found for investigating the function of the central processes in audition many of the conclusions arrived at through use of the Wever and Bray technic may have to be modified.

This hypothesis is based on the following evidence:

1. Hearing is never demonstrable in the absence of the cochlear response.

2. Cochlear potentials are absent in deaf animals.

3. Cochlear potentials faithfully reproduce in a loud speaker the actual sounds made into the subject's ear.

4. Equal potentials are produced at different frequencies by the same strengths of stimulation as yield tones rated equal in loudness by human observers.[24]

SUMMARY

Experimentation related to the nature and the function of the electrical response of the cochlea to acoustic stimuli is reviewed, and the following conclusions are arrived at:

1. The Wever and Bray phenomenon as originally observed consists of two distinct components: first, an electrical spread from the cochlea, and second, action currents from the auditory nerve.

2. The characteristics of each component render it easily distinguished from the other.

3. The origin of the cochlear response is found in the organ of Corti.

4. The probable process of generation is a piezoelectric effect created by the mechanical distortion of the hair cells of Corti's organ caused by the sound waves impinging on the tympanic membrane.

5. On the basis of the evidence presented, the cochlear response is regarded as an index of hearing with certain limitations placed on interpretation by the lack of knowledge of central processes.

McGill University Faculty of Medicine.

40. Lorente de Nó, R.: The Reflex Contractions of the Muscles of the Middle Ear as a Hearing Test in Experimental Animals, Tr. Am. Laryng., Rhin. & Otol. Soc. **39**:26-42, 1933.

41. Covell, W. P., and Black, L. J.: The Cochlear Response as an Index of Hearing, Am. J. Physiol. **116**: 524-530, 1936.

BDURAL EMPYEMA SECONDARY TO PURULENT FRONTAL SINUSITIS

A CLINICOPATHOLOGIC STUDY OF FORTY-TWO CASES VERIFIED AT AUTOPSY

CYRIL B. COURVILLE, M.D.

LOS ANGELES

The presence of purulent exudate in the cranial bdural space, a condition which has been signated by a variety of names, has been recog- ed as a pathologic entity for a little more than f a century. The term "subdural abscess" was d to introduce and describe a relatively rare d for the most part clinically unimportant mplication of purulent otitis media. For some known reason, relatively little attention has en paid to the more common, more easily cognized and therapeutically much more im- rtant subdural empyema consequent to purulent ntal sinusitis. As close a student of the oblem as Gerber was content to dismiss the bject in 1909 with less than a page of his onograph concerned with complications of ntal sinus infections. But it seems as though en this suggestion that the purulent subdural cumulation might be a common complication frontal sinus disease (found six times in rber's series of 21 cases) seems to have been gely lost sight of clinically. Today one searches most in vain for mention of the possibility of is complication either by a pathologist or by a nician. Most textbooks of neuropathology are o strangely silent on this significant and fairly mmon consequence of purulent frontal sinusitis. Perhaps because of its relatively frequent currence as an intracranial lesion, as discovered a rather large series of autopsies, I have come especially interested in subdural em- ema as a pathologic entity, and frequent ention has been made of it in a series of studies uing from the Cajal Laboratory of Neuro- thology which have been separately concerned ith its classification and morbid anatomy,[1] its cidence as a complication of otitis media and mastoiditis,[2] its development consequent to rupture or surgical drainage of a cerebral or a cerebellar abscess,[3] its occurrence as a complication of frontal sinusitis[4] and its development as a complication of cranial trauma.[5]

It is the purpose of this study to present specifically the characteristics of subdural empyema (or abscess) consequent to purulent infection of the frontal sinus, which produces in many cases a well defined symptom complex that makes possible a correct diagnosis. Since in some cases the pathologic process is curable by surgical intervention, it is to be hoped that the present high mortality will be remarkably reduced in the next few years.

TERMINOLOGY

A survey of the various possible inflammatory lesions which may involve the subdural space as complications of purulent frontal sinusitis makes it clear that no one title is comprehensive enough to include them all. Among the many designations which have been used to describe them are "pachymeningitis interna," "intradural abscess," "intrameningeal abscess," "subdural abscess" and "subdural empyema." Since the term "pachymeningitis interna," strictly speaking, applies to the less conspicuous diffuse inflammatory reaction without gross formation of pus and obviously of little clinical interest, this term is rejected as impracticable. Of the other terms which indicate grossly evident collections of pus in the subdural space, "subdural abscess" and "subdural empyema" have been selected as the most descriptive. From a pathologic point of view "subdural abscess" might best be reserved for the more circumscribed, well walled-off accumulation of pus observed in chronic cases, while "subdural empyema" might

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1. Courville, C. B.: Pathology of the Central ervous System, Mountain View, Calif., Pacific Press ublishing Association, 1937, pp. 112-116.

2. Courville, C. B., and Nielsen, J. M.: Fatal mplications of Otitis Media, with Particular Refer- ce to the Intracranial Lesions, in a Series of Ten housand Autopsies, Arch. Otolaryng. **19**:451 (April) 34; Intracranial Complications of Otitis Media and astoiditis: Statistical Study with Survey of Ten housand Necropsies, Acta oto-laryng. **21**:19, 1934.

3. Courville, C. B., and Cutler, O. I.: Subdural Abscess Secondary to Surgical Drainage of a Cerebral Abscess, Bull. Los Angeles Neurol. Soc. **1**:114, 1936.

4. Courville, C. B.: Rhinogenous Subdural Abscess: Report of Two Cases, One with Associated Frontal Abscess, Bull. Los Angeles Neurol. Soc. **1**:55, 1936. Courville, C. B., and Rosenvold, L. K.: Intracranial Complications of Infections of Nasal Cavities and Accessory Sinuses, Arch. Otolaryng. **27**:692 (June) 1938.

5. Courville, C. B., and Blomquist, O.: Traumatic Pachymeningitis Interna and Subdural Abscess, Arch. Surg. **42**:890 (May) 1941.

more properly be applied to the more extensive and thin diffusion of exudate. However, since this distinction cannot always be made clinically, and since both terms are in common use, both will likely continue to be used to describe the clinicopathologic complex which may now be recognized as a clearcut entity.[6]

It is the avowed purpose of this study to establish in the minds of rhinologists and neurologists alike that subdural abscess secondary to suppurative frontal sinusitis ("empyema of the frontal sinus") is a well defined clinicopathologic entity which can be correctly diagnosed, appropriately treated and sometimes cured. It is for the same purpose that this study is entitled "subdural empyema secondary to frontal sinusitis" rather than the more inclusive "rhinogenous subdural abscess." And this title is used in spite of the fact that in some cases pansinusitis was present, for it was clear that the extension to the subdural space had its origin in the frontal sinus rather than in any of the other sinuses which may have been coincidentally affected. The extremely rare but possible subdural abscess secondary to purulent rhinitis in infants and young children very likely has a similar (vascular) causation even though the frontal sinus is rudimentary. Only when these fundamentals are kept in mind can the ultimate purpose of a study such as this be achieved, i. e., to save the life of a patient otherwise doomed to an untimely death.

ANATOMY OF THE SUBDURAL SPACE

Like the other mesothelium-lined spaces, the subdural space in the living person is a potential one only, for the brain and its system of blood vessels, its membranes and the enclosed cerebrospinal fluid completely fill the cranial cavity. The brain, however, occupies this space loosely. It is anchored at the base by the cranial nerves in their passage through the foramens, by the infundibulum of the hypophysis, by the meningeal arteries and the accompanying veins, by the medulla as a continuation of the spinal cord and by the internal carotid and vertebral arteri at their entrance. It is suspended at the vert by the superior cerebral veins at their entran and the attachments of the pacchionian granul tions; it is less securely held posteriorly a below by the inferior cerebral and inferi cerebellar veins at their exits and the great ve of Galen, which empties into the straight sinus a point where the inferior margin of the fa cerebri joins the anterior margin of the te torium cerebelli.

The more intimate relation of the irregul base of the brain with the fossae of the sk as well as the firm attachment to the cranial ba by means of the cranial nerves, makes the cavu subdurale in this region a space in name only.

Accumulations of foreign material in th region are always small, too small to be of an clinical importance as far as their mass is co cerned. A different situation, however, exis over the dorsolateral surfaces of the cerebr hemispheres. Here the space extends wide without anatomic interruption, which favors th accumulation of large amounts of spinal flui blood or purulent exudate, sufficient at times depress seriously the underlying cortex and dislocate the entire cerebral hemisphere and th enclosed lateral ventricle.

The cavum subdurale is lined with flattene mesothelial cells, one layer forming the lini layer of the membranous structure, the dur mater, and the other forming the external laye of the arachnoid. In the normal state these tw layers are everywhere in contact. It is from th mesothelial cells lining this cavity that the ne membrane which envelops any collection of cere brospinal fluid, blood or pus is formed.

HISTORIC CONSIDERATIONS

The recognition of subdural inflammator lesions in their various manifestations and com binations is by no means a modern achievement Undoubtedly instances of these lesions were de scribed in the European literature of the eighteenth century, now largely unavailable in this country

The first suggestive case was that of Richter reported in 1773. His patient, a man of 50 years had a difficulty of speech and right hemiparesi that developed consequent to chronic frontal sinu sitis. He died in coma about five days after th onset of swelling over the left eye. A complet autopsy was not made, but exploration of th frontal sinus disclosed a perforation of the pos terior wall. Further exploration with a soun disclosed pus within the intracranial space, ap parently recovered from the subdural space.

6. It is not only the problem of the nature and the extent of the inflammatory reaction which makes exact designations difficult but also the etiologic problem, which further tends to complicate the matter. For the purpose of simplicity and that of practicality from a clinical standpoint, it seems desirable to classify such lesions on the basis of the source of infection rather than to lump together all cases of empyema of the subdural space. This is particularly true because subdural abscess secondary to frontal sinusitis is a relatively common, fairly readily discoverable and easily localizable lesion, while the same lesion with the same symptom complex secondary to otitis media is less common and seldom assumes a size to be of clinical significance. Moreover, the traumatic lesion is rare although also of clinical import. Those of any other causation are clinical rarities and pathologic curiosities.

7. Richter, G. A.: De morbis sinuum frontal, 177 cited by Dreyfuss,[19] pp. 13, 64 and 102.

The first case of possible subdural abscess ified at autopsy was studied in 1799 and orted in 1812 by Schütz,[8] who found a strik- inflammation of the surface of the left frontal e of the brain, evidently secondary to purulent ntal sinusitis. The extremely rare develop- nt of leptomeningitis as a direct consequence purulent frontal sinusitis suggests that this ion was actually a subdural inflammatory cess. In a case reported in 1806 by Celliez[9] s was recovered from the subdural space by ssage of a sound (at autopsy) through an flammatory defect in the posterior wall of the ntal sinus. The patient had convulsions prior her death.

In Paulsen's[10] case (1861) the left cerebral misphere was compressed by 8 ounces (236.5) of thick pus in the left "cavum arachnoid." ere was a fistula through the bone and the ra to the accumulation of pus in this region. ree days before death the patient had pre- nted paralysis of the right side of the body. seems clear that this was a true subdural scess and not localized leptomeningitis, a con- sion shared by Gerber.

The case reported by Bourot and Lécard[11] 875) is very likely one of unilateral subdural scess, although these writers described the tracranial lesion as a "meningite partielle con- cutive" (secondary localized meningitis). The ason for believing that this was in fact a sub- ral and not a subarachnoid collection of pus that the patient, a 24 year old soldier, had ft hemiplegia following what seems to have en acute suppurative frontal sinusitis with tension into the adjacent orbit. Hemiplegia is rare complication of meningitis but is common ter subdural collection of pus, being due to ressure on the corresponding motor strip. The meningite" was evidently one sided, on the ght, the same side as the presumed empyema of e frontal sinus.

Bosquet[12] (1877) in his case found the surface f the frontal lobe of the brain to be discolored a "dirty gray," evidently covered with exudate. A fistula connected the right frontal sinus and the anterior fossa of the skull. There was an encapsulated abscess the size of a hen's egg in the frontal lobe. In 1882 Huguenin[13] discussed what seems to be the first case of chronic pachymeningitis interna secondary to chronic frontal sinusitis. There was found at autopsy a fusion of the dura, the pia and the cortex, "the entire binding connective tissue was infiltrated with pus."

In Carver's[14] case (1883) the frontal convolutions were covered with exudate and the superior longitudinal sinus was filled with dirty gray pus. This is one of the first cases in which the association of thrombosis of the superior longitudinal sinus and subdural abscess was discovered. In the case reported by Schäfer,[15] in 1883, both subdural and intracerebral abscesses were described as a consequence of empyema of the frontal sinus. The case of Browne[16] (1893) was still another classic example of a rhinogenic subdural abscess verified at autopsy. That same year in his now memorable monograph Macewen[17] described briefly Miller's case of subdural abscess secondary to frontal sinusitis.

The case of Wallenberg[18] (1895) is generally accepted as one of a suppurative subdural lesion of frontal sinus origin. The patient, a 52 year old woman, was the victim of chronic sinusitis. The most recent episode was ushered in by two epileptiform convulsions, an exaggeration of her headaches and ptosis of the left upper eyelid. Before death she had motor aphasia followed by right hemiparesis. She died following exploration of the frontal sinus (at which time necrosis of the posterior wall of the frontal sinus and regional discoloration of the dura were discovered) and puncture of the brain. At autopsy a considerable amount of pus was found in the left subdural space.

The first available monograph dealing with the intracranial complications of disease of the

8. Schütz: Stirnhöhlenabscess, Allg. med. Ann., 1812, 750; cited by Kühnt,[20] p. 150.

9. Celliez: Observation sur un abscès dans le sinus ontal et dans le crâne, accompagné de la paralysie de paupière supérieure, et d'autres symptomes très re- arquables, J. de méd., chir., pharm. **11**:516, 1806; ted by Kühnt,[20] p. 150.

10. Paulsen, J. P., 1861, cited by Kühnt,[20] p. 150.

11. Bourot and Lécard: Observation d'un phlegmon e l'orbite droite avec meningite partielle consécutive; ort dans le collapsus et le coma au cinquième jour du raitement. Autopsie, réflexions; cited by Kühnt[20] and erber.[24]

12. Bosquet: Abscès developpé dans le sinus frontal u côté droit; collection purulente dans le lobe frontal u même côté; trepanation; mort, Bull. Soc. anat. de aris **2**:512, 1877.

13. Huguenin: Zur Casuistik der Meningitiden, Cor.-Bl. f. schweiz. Aerzte **12**:97, 1882.

14. Carver: Acute Necrosis of the Orbit, Brit. M. J. **1**:1183, 1883.

15. Schäfer, H.: Ein Fall von ulceröser Entzündung in Bereiche der rechten Siebbenhälfte mit letalem Ausgange, Prag. med. Wchnschr. **8**:189, 1883; cited by Kühnt,[20] p. 161.

16. Browne, L.: A Case of Suppuration of the Frontal Sinus, J. Laryng., Rhin. & Otol. **7**:53, 1893.

17. Macewen, W.: Pyogenic Infective Diseases of the Brain and Spinal Cord. Glasgow, James Maclehose & Son, 1893.

18. Wallenberg: Empyem des linken Sinus frontalis: Perforation nach Schädelhöhle hin, intradurale Eiterung, Neurol. Centralbl. **14**:903, 1895; cited by Gerber,[24] p. 290.

accessory nasal sinuses is that of Dreyfuss.[19] In the section on intracranial complications of frontal sinusitis, the cases herein mentioned are described in some detail, although Dreyfuss made no clearcut distinction between intracerebral and subdural abscess. This study exemplifies the difficulty of attempting to evaluate the descriptions of the lesions verified at autopsies in this group of older cases. Some of the lesions described are at best only presumptive examples of the type of lesion under consideration here.

Apparently the first monograph to deal with the problem of inflammatory diseases of the frontal sinuses and their complications was that of Kühnt,[20] which was published in 1895. He reviewed the literature on cases of empyema of the frontal sinuses up to that time, which cases were described in extenso. Like so many of those who followed him in this field of investigation, Kühnt was more concerned with the genesis of intracranial complications, particularly of abscess of the frontal lobe, than he was with subdural abscess. This may have been due in part to the fact that in none of his personally studied cases was this complication found.

In 1900 Killian,[21] reviewing the literature for instances of thrombosis of the superior longitudinal sinus resulting from frontal sinusitis, described 5 cases, in 2 of which (that of Macewen and Miller and that of Miller) subdural abscesses were found. Hajek[22] (1906), reviewing the general problem of intracranial infections complicating disease of the accessory nasal sinuses, discussed the genesis of subdural abscess, among other lesions, without reporting any personally studied cases. Two years later Dreyfuss[23] reviewed the literature on intracranial complications of infections of the accessory nasal sinuses. A total of 91 cases of complications of frontal sinusitis were studied, and the pathway of infection was apparently recognized in 88. Only 5 cases of subdural abscess were mentioned as having been reported; many of the cases cited in contemporary literature must have been misinterpreted or overlooked.

It remained for Gerber[24] (1909) to make a critical review of the subject of frontal sinusitis and its intracranial complications. He surveyed the findings at operation and autopsy in a large number of cases, but in only 6 cases was the term "subdural abscess" specifically used according to his own summary. However, in his *Kasuistiks* of the various categories, what seem to be 11 unquestioned cases of acute or chronic subdural inflammatory lesions are described (Celliez, 1806; Paulsen, 1861; Huguenin, 1882; Browne, 1893; Macewen and Miller, 1893; Silex, 1896; Blessig-Tiling, 1898; Trautmann Stenger, 1900; Reinhard, 1905; Claoue, 1906). Under the heading of "pachymeningitis interna, intradural, intrameningeal abscess, subdural abscess" he simply stated the fact that uncircumscribed accumulations of pus may be found between the "leaves" of the dura or between the dura and the pia, involving the inner surface of the former. Thus by 1909 the possibility of the occurrence of subdural infectious lesions with origin from infection of a frontal sinus was definitely established.

In 1913 McKenzie[25] published his well known paper on osteomyelitis of the skull secondary to inflammatory diseases of the nasal sinuses, having collected from the literature a series of 48 cases. In 3 cases subdural abscess was found.

In 1923 Toti[26] published a rather extensive review of all cases of intracranial complications of disease of the accessory nasal sinuses reported since the appearance of Gerber's monograph in 1909. With his 267 collected cases added to those of Gerber a grand total of 530 cases was available for analysis. Unfortunately, the omission of details made it difficult to distinguish between extradural and subdural suppurative lesions in the 19 cases of pachymeningitis in the 134 instances of complications of frontal sinusitis.

In a relatively recent survey of 36 cases of rhinogenous intracranial complications which came to light at the Municipal Hospital in Copenhagen between 1906 and 1933, Hagerup[27] briefly described 4 cases of subdural abscess.

19. Dreyfuss, R.: Die Krankheiten des Gehirns und seiner Adnexa im Gefolge von Naseneiterungen, Jena, Gustav Fischer, 1896, pp. 12-29 and 64-67.

20. Kühnt, H.: Ueber die entzündlichen Erkrankungen der Stirnhöhlen und ihre Folgezustände, Wiesbaden, J. F. Bergmann, 1895.

21. Killian, G.: Die Thrombophlebitis des oberen Längsblutleiters nach Entzündung der Stirnhöhlenschleimhaut, Ztschr. f. Ohrenh. **37**:343, 1900.

22. Hajek, M.: Ein Beitrag zum Studium des Infectionsweges bei der rhinogenen Gehirnkomplikation, Arch. f. Laryng. u. Rhin. **16**:290, 1906.

23. Dreyfuss, R.: Rhinogene Gehirnaffektionen, Internat. Zentralbl. f. Ohrenh. **6**:103, 1907.

24. Gerber, P. H.: Die Komplikationen der Stirnhöhlenentzündungen: Beiträge zur Anatomie und Klinik der Stirnhöhlen, Berlin, S. Karger, 1909.

25. McKenzie, D.: Diffuse Osteomyelitis from Nasal Sinus Suppuration, J. Laryng., Rhin. & Otol. **28**:6 and 129, 1913.

26. Toti, E.: Kasuistik der intrakraniellen Komplikationen akuter und chronischer Nebenhöhlenerkrankungen seit den Arbeiten von Gerber und Dreyfuss bis zum Jahr 1923, Ztschr. f. Hals-, Nasen- u. Ohrenh. **7**:41, 1923.

27. Hagerup, G.: Rhinogenous Intracranial Complications, on Basis of Material of Department for Diseases of the Ear of Municipal Hospital, Copenhagen, Acta oto-laryng. **24**:321, 1936.

A recent contribution to the subject of subdural empyema in general by Kubik and Adams [28] includes a summary of 12 cases of this condition secondary to paranasal sinus infection. In 2 cases recovery followed surgical drainage. Kubik and Adams concluded that chronic rather than acute sinusitis with frontal osteomyelitis and subdural infection was particularly favorable to the development of subdural empyema.

A complete survey of the literature in the hope of analyzing each case in detail is almost impossible. Many of the earlier cases are described in local publications not available in this country. In many reports desirable details are wanting. Furthermore, Toti in a review which completed up to 1923 the surveys of Gerber and Dreyfuss did not abstract the individual cases which made up his totals as his predecessors had done. It is therefore impossible to know just what cases are referred to in his final figures. While a search has been made in available literature for cases which might be included in articles dealing with frontal sinusitis and its intracranial complications, many instances have likely been overlooked. Thus far, I have been able to find only 48 reasonably well defined cases of subdural abscess secondary to nasal sinusitis. This must represent only a small fraction of those which are actually verified and perhaps even reported under nonsignificant titles. Still, this total corresponds quite well with the 42 cases referred to by Dreyfuss in 1908, the 6 cases mentioned by Gerber in 1909 and the 19 cases of "pachymeningitis" included by Toti in his review in 1923. The limitations of space forbid even a brief summary of any of these cases in this connection.

CASES PERSONALLY STUDIED

This study is based on a series of 42 cases of subdural abscess or empyema consequent to acute suppurative infection of the frontal sinus which were verified at autopsy. Cases verified at operation in which the patients were either cured or died without necropsy have been omitted since all the pathologic details of this entity were not available for study. Only by this selection was the complete and uninterrupted clinical course of the disease available for survey; in addition there was an opportunity to study the entire postmortem situation. It is to be hoped that once physicians become alert to the occurrence of the lesion and the method by which it can be cured, more cheerful reports on the incidence of cures will be forthcoming. With this, as with many other suppurative lesions, a full knowledge of its evolution and character must precede successful therapy. The important details of the cases on which this study is based are to be found in the accompanying table. The inclusion of full case reports with so large a series of cases is beyond the reasonable limitations of publication in a journal such as this.

PATHOGENESIS

As in cases of otitic intracranial complication, the pathway traveled by micro-organisms from the frontal sinus to the subdural space to produce a subdural infection is variable and not always distinguishable at autopsy. In some cases it is necessary to presume that the invasion has occurred by way of vascular channels, since no other path is evident. Direct extension through the posterior wall of the frontal sinus, made possible by the development of a focus of infectious osteitis and subsequent erosion through the contiguous dura, is the most direct and obvious mode of spread. It is comparable to extension through the tegmen tympani and regional dura in the case of otitic infection. The route suggested is not often followed in spread of subdural infection from the frontal sinus, however, for the discovery of a bone-dura fistula between the frontal sinus and the subdural space is a rarity. Theoretically, surgical enlargement of such a channel would result in evacuation of the underlying abscess, but such an eventuality has never been recorded so far as I know.

Indirect bony extension, through the intervention of a more extensive osteomyelitic process, may also account for spread of infection to the subdural space. In such an event an extradural abscess is likely to develop as an intermediate lesion. From this extradural focus the infection may extend through the dura by formation of a fistula, or more commonly it may spread by way of small venous radicals, or, finally, it may reach the subdural space by concomitant extension through the superior longitudinal sinus and its afferents, the superior cerebral veins.

The pathway of the communicating veins is perhaps the most common of all avenues of subdural infection. This is suggested by the fact that in the majority of cases coming to autopsy no grossly visible changes are found in bone or dura which might account for the development of subdural empyema. This route is clearly suggested in those cases in which the superior longitudinal sinus and the superior cerebral veins show well developed thrombosis as an obvious predisposing condition. Whether the infection travels from the small veins of the membranous lining of the frontal sinus directly into

28. Kubik, C. S., and Adams, R. D.: Subdural Empyema, Brain **66**:18, 1943.

Cases of Subdural Empyema Secondary to Frontal Sinusitis ("Rhinogenous Subdural Abscess") Verified at Autopsy (Cajal Laboratory Series)

No.	Lab. No.	Age	Sex	Sinusitis	Survival Period *	Location of Subdural Empyema	Associated Lesions	Localizing Symptoms and Signs
1	5650	40	♂	Acute right frontal	2 weeks ?	Dorsolateral surfaces of left frontal and parietal lobes	Osteomyelitis of frontal bone, extradural abscess, ulceration of tip of right temporal lobe	Enlarged right pup
2	6143	12	♂	Acute left frontal	4 to 6 days	Dorsolateral surfaces of left frontal and parietal lobes	None	None observed
3	6464	9	♂	Acute right and left frontal	3 days	Dorsolateral surfaces of right frontal and parietal lobes	Bone-dura fistula in posterior wall of right frontal sinus	None observed
4	7016	50	♀	Acute right frontal	2 to 3 days	Dorsolateral surfaces of both frontal lobes	Focal erosion in posterior wall of right frontal sinus; meningitis	None observed
5	8561	14	♂	Acute left frontal	10 days ?	Dorsolateral surfaces of both frontal lobes	Subaponeurotic abscess, osteomyelitis of frontal bone, extradural abscess	None observed
6	9050	17	♂	Acute left frontal	3 days	Dorsolateral surface of left frontal lobe	Extradural abscess, thrombosis of superior longitudinal sinus	Generalized convulsions
7	9656	47	♂	Subacute pansinusitis (right and left frontal)	2 weeks ?	Dorsolateral and basilar surfaces of right cerebral hemisphere	Extradural abscess, thrombosis of superior longitudinal sinus and veins, focal red softening of regional cortex	Alternating spastic and flaccid left hemiplegia
8	9723	53	♂	Acute left frontal	2 weeks	Dorsolateral surface of left cerebral hemisphere	Extradural abscess, thrombosis of superior longitudinal sinus, multiple small abscesses of left frontal lobe	Spastic right hemiparesis, with increase of deep reflexes on right
9	10-116	37	♀	Acute right frontal	3 to 5 days	Dorsolateral, mesial and basal surfaces of right frontal lobe	Internal hydrocephalus, meningitis	None observed
10	10-225	52	♂	Chronic right and left frontal	3 days	Dorsolateral surfaces of right cerebral hemisphere and left frontal lobe	Infection of scalp, osteomyelitis of frontal bone, extradural abscess, edema of right frontal lobe	Right jacksonian convulsions with increase of deep reflexes and pathologic reflexes on right
11	12-407	33	♂	Acute left frontal	3 days	Dorsolateral surfaces of right frontal and parietal lobes	None	Spastic weakness of right arm, increased deep reflexes and pathologic reflexes on right
12	12-661	27	♂	Chronic recurrent right frontal	2 weeks ?	Dorsolateral surfaces of right frontal and parietal and left frontal lobes	Osteomyelitis of frontal bone, extradural abscess, meningitis	Focal convulsions of left side of face, left flaccid hemiparesis
13	14-681	24	♀	Acute pansinusitis (right and left frontal)	5 days	Dorsolateral surfaces of left frontal and parietal lobes	Subaponeurotic abscess, focal necrosis of bone, extradural abscess, meningitis	Convulsive movements of right side of face and arm, right hemiparesis, motor aphasia
14	14-761	27	♀	Acute left frontal	1 week ?	Dorsolateral surface of left cerebral hemisphere	Erosion of posterior wall of left frontal sinus, extradural abscess, thrombosis of superior longitudinal sinus, small abscess of left frontal sinus	Jacksonian convulsions of left side, generalized convulsions, left hemiparesis
15	15-263	29	♂	Acute pansinusitis (right frontal only)	5 to 8 days	Dorsolateral surface of right frontal lobe	Meningitis	Transitory weakness of lower part of left side of face
16	15-313	11	♀	Acute pansinusitis (right and left frontal)	20 days ?	Dorsolateral surfaces of both frontal lobes, more on left side	Meningitis	Transitory weakness of lower part of right side of face
17	15-963	17	♂	Acute right frontal	3 days	Dorsolateral surfaces of both frontal lobes, more on right side	Subaponeurotic abscess, local meningitis	None observed
18	16-206	16	♂	Acute left frontal	4 days ?	Dorsolateral surfaces of left frontal and parietal and right frontal lobes	Subaponeurotic abscess, extradural abscess, intrasinus abscess, cortical softenings (multiple)	Hypotonia on left, transitory Babinski sign on left
19	17-498	16	♀	Acute left frontal	14 days	Dorsolateral and mesial surfaces of left cerebral hemisphere	Extradural abscess, thrombosis of superior longitudinal sinus, small abscess of left frontal lobe, meningitis	Spastic weakness of right arm, motor aphasia
20	17-696	13	♂	Acute right frontal	3 weeks +	Dorsolateral surface of right frontal lobe	Osteomyelitis of frontal bone, extradural abscess, large abscess of right frontal lobe	Hemiparesis on left

Cases of Subdural Empyema Secondary to Frontal Sinusitis ("Rhinogenous Subdural Abscess") Verified at Autopsy (Cajal Laboratory Series)—Continued

Lab. No.	Age	Sex	Sinusitis	Survival Period *	Location of Subdural Empyema	Associated Lesions	Localizing Symptoms and Signs
18-417	16	♂	Acute right frontal	1 day +	Dorsolateral surface of right frontal lobe	Early focal erosion of bone, extradural abscess	Focal seizure on left side of face followed by preterminal generalized convulsions
14-494	12	♂	Acute right frontal	2 to 3 days ?	Dorsolateral surfaces of both frontal lobes	Meningitis	None observed
19-636	38	♂	Pansinusitis (?) (right and left frontal)	?	Dorsolateral surface of left cerebral hemisphere	Bone-dura fistula, extradural abscess	Complete motor aphasia
20-184	10 mo.	♀	Acute right and left frontal	11 to 12 days	Dorsolateral surfaces of both frontal lobes	Thrombosis of superior longitudinal sinus, red softening of frontal cortex	Jacksonian seizures, first on left then on right
21-230	25	♂	Acute pansinusitis (right and left frontal)	2 days +	Dorsolateral surface of left cerebral hemisphere	None	Suggestive motor aphasia
21-760	11	♂	Acute pansinusitis (left frontal)	5 days	Dorsolateral surfaces of both frontal lobes	Infection of scalp	Babinski sign and ankle clonus on left
23-591	15	♂	Acute right and left frontal	6 days	Dorsolateral and basilar surfaces of right frontal lobe	.....	Left pupil dilated, generalized convulsions
23-817	3 mo.	♂	Acute pansinusitis	?	Dorsolateral surfaces of both frontal lobes	Thrombosis of superior longitudinal sinus, meningitis	None observed
24-336	16	♀	Acute right frontal	2 to 3 weeks	Dorsolateral surface of left frontal lobe	Infection of scalp, extradural abscess, early meningitis	None observed
26-072	50	♂	Chronic recurrent pansinusitis (right and left frontal, more right)	3 days	Dorsolateral surface of left cerebral hemisphere	Foci of red softening of cerebral cortex	Motor aphasia, Babinski sign on right, right knee jerk greater than left
26-604	26	♂	Acute (?) right and left frontal	7 to 10 days	Dorsolateral and mesial surfaces of left frontal and parietal lobes	None	Right spastic hemiparesis, right homonymous hemianopsia
28-849	28	♀	Acute left frontal	2 days	Dorsolateral surface of left cerebral hemisphere	None	None observed
30-186	25	♂	Acute left frontal	7 days	Dorsolateral and mesial surfaces of left cerebral hemisphere	Extradural abscess thrombosis of superior longitudinal sinus, small distant (occipital) cortical abscesses	Jacksonian convulsions on right, right hemiparesis
30-578	23	♂	Chronic recurrent right frontal	4 days	Dorsolateral surface of right cerebral hemisphere	Early meningitis (?)	Left hemiparesis and hemihypesthesia
M-106	14	♀	Acute right frontal	Several mo.	Dorsolateral surface of right cerebral hemisphere	Old osteomyelitis of skull, adhesions and old cerebral abscesses (6)	Jacksonian convulsions on left, pathologic reflexes on left
M-235	8	♂	Acute left frontal	1 day ?	Dorsolateral surfaces of right and left frontal lobes	Extradural abscess	Generalized convulsions
M-328	15	♀	Acute right frontal	2 yr.	Dorsolateral surface of right cerebral hemisphere	Old osteomyelitis of skull, adhesions and old cerebral abscesses	Jacksonian convulsions on left
NC-76	13	♂	Acute right frontal	2 weeks	Dorsolateral surfaces of both cerebral hemispheres	Local erosion of bone, extradural abscess, thrombosis of superior longitudinal sinus, multiple cerebral abscesses	Poorly defined bilateral localizing motor manifestations
NC-306	41	♂	Acute left frontal	4 days	Dorsolateral surfaces of left cerebral hemisphere and right frontal lobe	Subaponeurotic abscess, osteomyelitis of frontal bone, extradural abscess	Right spastic hemiparesis and hemihypesthesia, convulsions (jacksonian on right and generalized), with deviation of head and eyes to left, motor aphasia
M-501	15	♂	Acute right and left frontal	4 days	Dorsolateral surface of right cerebral hemisphere	Focal erosion of bone, extradural abscess	None recorded
NC-A1	15	♀	Acute right and left frontal	2½ weeks	Dorsolateral surface of right cerebral hemisphere	Focal erosion of bone (?), extradural abscess	Flaccid left hemiplegia, facial twitchings on left
NC-A2	8	♂	Acute left frontal	3 days	Dorsolateral surface of right cerebral hemisphere	Erosion of posterior wall of left frontal sinus	Slight weakness of right arm

* This is the estimated period of survival with subdural empyema not with the causative frontal sinusitis.

the regional dural veins and thence into the superior longitudinal sinus or whether it extends first into the veins of the diploic spaces is a matter of theoretic interest only.

There are a number of questions which this problem of venous extension brings up which cannot be answered with certainty. Why does this extension by the venous route result in subdural localization of infection (empyema) rather than in subarachnoid contamination (purulent meningitis), which seems to be a rare direct and isolated consequence of such invasion? Why does subdural localization occur more frequently and more abundantly than localization within the brain substance with the formation of an abscess? The answers to these questions are probably to be found in the peculiar manner in which the superior cerebral veins expose themselves in the subdural space in the course of their debouchment into the superior longitudinal sinus and perhaps in the chance character of the ultimate venous lesion. Thrombosis of the smaller venous radicals with consequent cortical softening would tend to pave the way for formation of a frontal abscess, while direct infiltration of the organisms into the subdural space would more likely result in a subdural abscess. Not infrequently both types of invasion occur, and multiple small circumscribed but not well encapsulated cortical and subcortical abscesses may be found as lesions coincident with the subdural collection of pus.

Under any circumstances the evolution of the subdural empyema tends to be remarkably rapid. When one views the extent of the lesion and the amount of pus present in it at the acute stage in the light of the relatively few days in which the lesion has had time to develop, the conclusion is evident that the reactive inflammatory response to the invasion is prompt and effusive. There must be wide variations in the virulence of the organism and the degree of bodily resistance to account for the variability of the fluidity and the amount of free pus, the limitations of its spread and the degree of encapsulation. The relative infrequence of development of the subacute or chronic state (chronic adhesive pachymeningitis interna), the early high mortality and the rarity of secondary serious complications make it clear that subdural empyema is a lethal lesion in its own right and one which demands a great deal more attention than it has received in the past.

The course of the lesion, once developed, is a matter of considerable interest from a pathologic as well as from a therapeutic standpoint. In addition to whatever serologic and cytologic resistance to the infection may be put forth there occurs a distinct effort to limit the spread of infection by deposit of fibrin and formation of a neomembrane. This formation of a neomembrane is apparently the typical subdural reaction to all foreign materials whether pus, free blood or cerebrospinal fluid. The flattened cells of the membrane seem to come from the dura itself. This effort at localization of the abscess, however, is rarely effective, and the exudate tends to spread in the subdural space until the toxemia incident to the mass of the pus, aided and abetted by the increasing intracranial pressure and developing meningitis, closes the case once and forever.

At times, however, some temporary limitation of the process seems to occur. In some cases more sharply defined circumscription of the purulent process is evident on the surface of the brain. Within the limits of this protective barricade is evident the depression which represents the site of accumulated pus. Break-throughs are sometimes evident and secondary accumulations, usually posterior to the primary focus, may likewise be at least partially circumscribed.

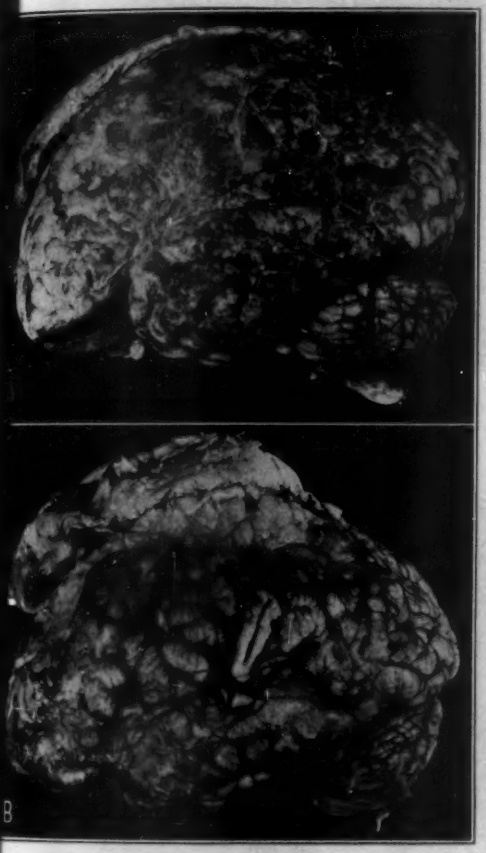
Extension of the pus seems to take place chiefly in a posterior direction, possibly aided by gravity (dorsal recumbent position of the patient). The anteroposterior extent of the exudate is usually greater than its vertical extent. The lower border of the area is fairly smooth contoured, suggesting an evenly progressive invasion of the subdural space. The pus tends to accumulate in the natural depression of the sylvian fissure whence it courses toward the basilar cisterns. The basilar surfaces of the frontal, and particularly of the temporal lobes, are rarely reached by the exudate. This is probably due to the fact that the basilar surface of each of these lobes is more closely approximated to the internal surface of the skull than is the dorsolateral surface.

MORBID ANATOMY OF SUBDURAL EMPYEMA WITH ORIGIN FROM FRONTAL SINUSITIS ("RHINOGENOUS SUBDURAL ABSCESS")

When one considers the prominence of this lesion here under consideration and its frequency in any large series of cases coming to autopsy, it is indeed remarkable that its identity has so long escaped the attention of pathologists, particularly of those who have given their special attention to the nervous system. But such seems to be the case. It is the object of this section of the essay to so describe subdural abscess with origin from frontal sinusitis as to make its character unmistakable—certainly to distinguish it from septic meningitis, with which it is often confused.

Character of the Exudate.—Subdural empyema (or abscess) secondary to frontal sinusitis

e defined as a gross accumulation of free the dorsolateral, basal or mesial subdural The exudate may be variable in color, ding on the organism which provokes its ction, yellow in consequence of an infection Staphylococcus aureus or variable tints of v-green if the infectious agent is Streptos haemolyticus or any one of the many of Pneumococcus. Its fluidity depends on mount of fibrin which it contains, which in may be a manifestation of the relative ence of the organism. In some cases there is



ig. 1.—Types of exudate in subdural empyema: *A*, watery exudate leaving little residue after fixa- *B*, heavy fibrinous exudate on the inner surface he dura and the outer surface of the leptomeninges.

y a small amount of very fluid exudate, and these cases there is little residual exudate nd on the fixed specimen (fig. 1 *A*). In eral the larger the amount of accumulated the more fluid it is and the less apt it is to limited by a neomembrane. Conversely, in ly cases or those with evolution the exudate y be quite plastic and more limited in extent amount (fig. 1 *B*). It is this type of lesion ich is most likely to become organized as onic pachymeningitis interna with localized dural collections of encapsulated pus scattered over the affected area; extension from lesions of this type accounts for "distant" abscesses of the brain.

The odor of the pus is not often disturbing, perhaps because of the absence of organisms usually responsible for foul odors (anaerobes) and because of the absence of decadent tissue, so often present in acute "stinking" abscesses of the brain.

As already intimated, the amount of pus differs greatly from one case to another. When pus is present in a liquid state, most of it escapes when the dura is cut. Therefore, the amount present in the subdural space can only be estimated. The largest recorded amount in any of the cases in this series was described by the pathologist as "about 100 cc." The amount evidently varies between a small amount of semifluid exudate, sufficient only to be scraped from the inner surface of the covering dura and the external surface of the subjacent arachnoid, up to amounts sufficient to flood the field when the dura is opened. The actual amount can be accurately estimated only when the lesion has been anticipated during life and precautions have been taken to collect it before the dura is widely incised. As a rule, the subdural space is evacuated at autopsy by accidental sawcuts of the dura before the intracranial space has been formally opened.

The best relative estimate of the amount of exudate, as well as of its limits, can be obtained from the degree and and the extent of the local depression of the cortex. When pus has been present in moderately large amounts, a well defined depression is evident in the fixed brain. Moreover, the greater the amount of pus the greater is the degree of distortion and dislocation of the ventricular pattern as visualized on coronal sections.

Distribution of the Pus.—The exudate occurs classically over the upper and anterior portions of the dorsolateral surfaces of the cerebral hemispheres (dorsolateral abscess). The upper portion of the frontal lobe is covered with a collection of exudate which usually extends over the parietal lobe as well. At times a second more or less distinct accumulation is found over this lobe (fig. 2). This second loculation is indicated by the presence of an independent depression in this region, which, while continuous with the first in its anterior and upper portion, is obviously not a part of it.[29]

29. One may be tempted to attribute this second collection of pus to an extension from the first, a "daughter" subdural abscess due to a break-through in the confining limiting membrane of the anterior col-

(Footnote continued on next page)

As a rule, the pus extends well down toward the curved inferolateral margin of the frontal lobe and into the sylvian fissure, which it may follow for variable distances posteriorly. It may also extend down this groove toward the base, thus accounting for the not infrequent exten of exudate into the chiasmal region. Rarely the pus extend down over the temporal co and when this incursion is present it is ordin limited in extent.

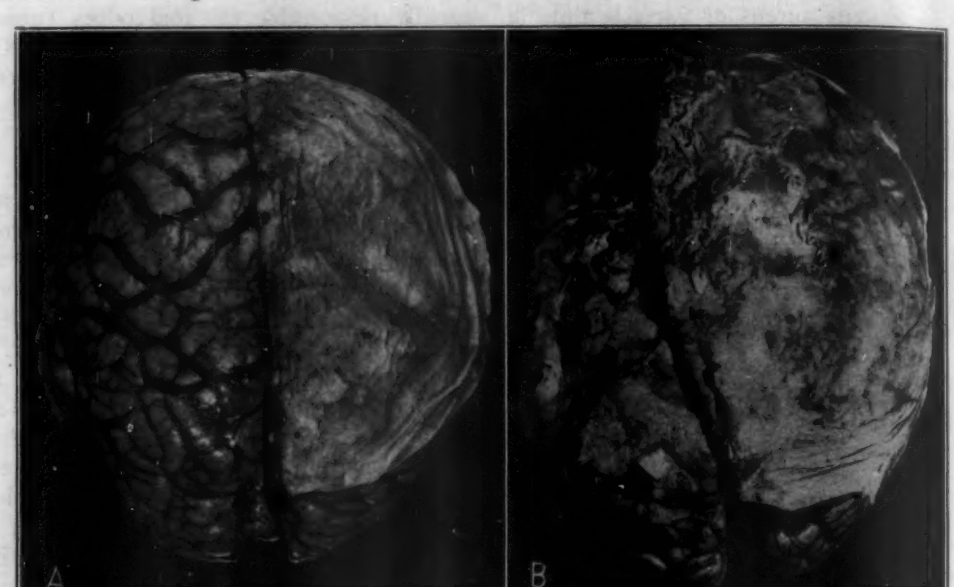


Fig. 2.—Distribution of exudate in subdural empyema (dorsolateral surface): *A*, diffuse spread of exu over the dorsolateral surface of the left cerebral hemisphere. *B*, localization of pus in two areas over the u surface of the hemisphere.

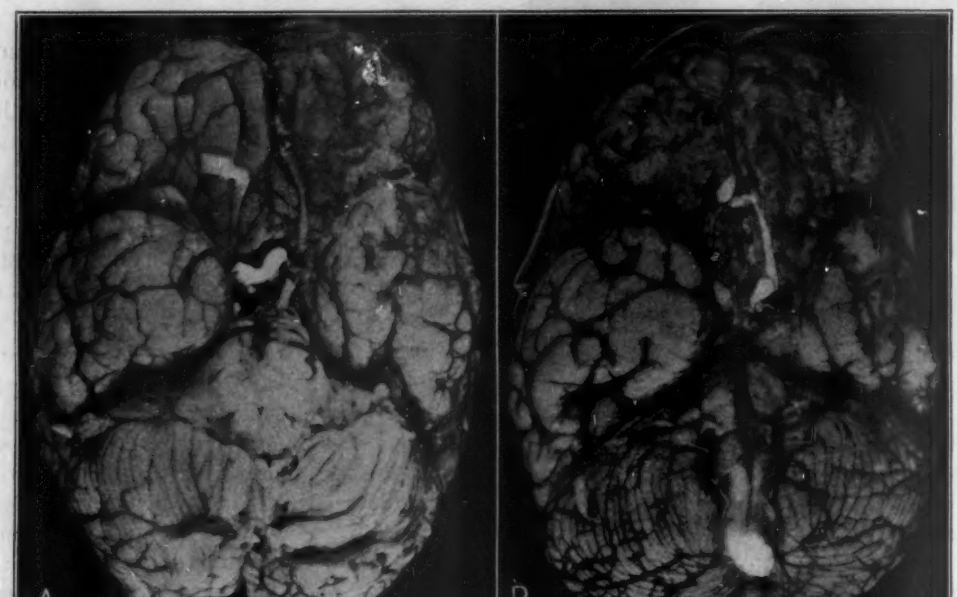


Fig. 3.—Distribution of exudate in subdural empyema (basilar surface): *A*, exudate on the basilar sur of the left frontal lobe and the tip of the left temporal lobe. *B*, bilateral subfrontal localization with additi exudate over the left temporal lobe.

lection. Such, however, does not seem to be the case. The absence of a limiting wall in this vicinity seems to exclude this possibility. Furthermore, one sees such double depressions with some examples of chronic subdural hematoma, suggesting that this phenomeno one incident to the collection of any fluid substanc the subdural space, an anatomic and mechanical ef rather than an etiologic one.

hen the pus is rather abundant (or in other ls, when the process has been one of fairly l evolution), pus may extend back onto dorsolateral surface of the occipital lobe, his is also unusual.

is likewise of less common occurrence for exudate to extend onto the basilar surface he frontal lobe, although this has been rved in the present series (fig. 3). Its rarity be explained by the intimate approximation e basilar surface of this lobe to the irregular convex surface of the orbital plate of the al bone. It is still more rare for the pus tend into the middle fossae of the skull, only such extension having been observed in this s. In this unusual case, however, the exudate found spreading out over the superior surface he tentorium and even into the posterior

e next most common region for the pus vade is the superior longitudinal fissure. mulating as the pus usually does in the r part of the dorsolateral subdural space, eeps through the intervals between the ers of pacchionian granulations and the hments of the superior cerebral veins to its way into this fissure. Here the pus cts in a variable amount depending on uidity and on the age of the lesion (fig. 4). e postmortem specimen the exudate is com- y found adherent to the medial aspect of rontal lobe and the opposed surface of the cerebri. Rarely pus is limited to the superior itudinal fissure (a mesial subdural abscess in adistinction to a dorsolateral subdural ess), constituting a definite clinical and ologic subtype. Only one such lesion was d in this series.

e extent of spread to the opposite hemi- re varies greatly from one case to another, nding on the amount of pus present, the of communication beneath the inferior free in of the falx cerebri [30] and perhaps the h of the survival period. Not infrequently ate is found on both sides of the falx cerebri topsy. In a smaller group of cases the pus und to have crossed over the supramesial r of the opposite hemisphere to reach the 's dorsolateral subdural space, the extension g place through the intervals between the s of granulations and the points of attach- s of the superior cerebral veins. Perhaps of the double or bilateral subdural abscesses o be accounted for by extension from one to the other by way of the superior longi-

tudinal fissure (fig. 5). In many cases they may well be due to simultaneous or concomitant involvement of both dorsolateral subdural spaces by way of the superior longitudinal sinus and the afferent superior cerebral veins (by retrograde extension).

Surgical Impressions of Lesion.—No description of the lesion under consideration would be complete without some note as to its surgical aspects. Through a trephine opening in the frontal bone the dura appears to have a lighter than normal color due to the presence of pus beneath this membrane. If the dura is now carefully opened, a new-formed membrane is often seen, appearing as a very thin, transparent, glistening tensile

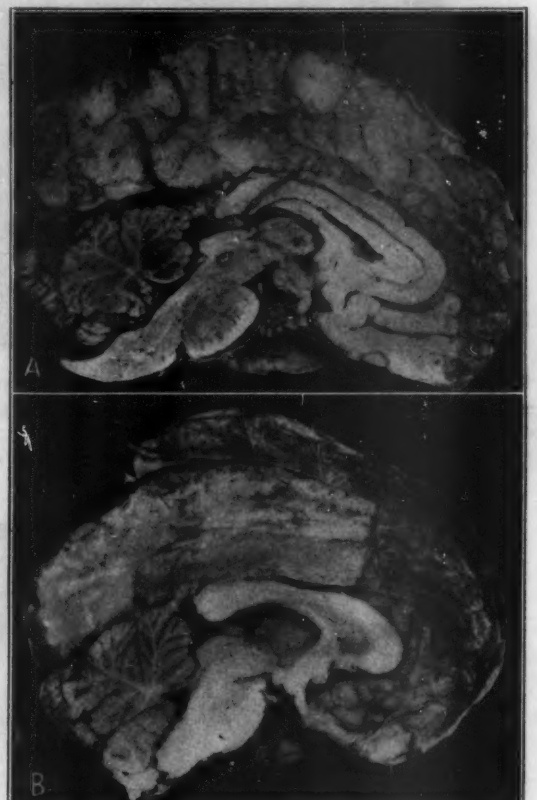


Fig. 4.—Distribution of exudate in subdural empyema (medial surface). *A*, exudate over the medial aspect of the left cerebral hemisphere. *B*, heavy loculation of exudate on the medial surface of the hemisphere.

covering which encloses the pus in the subdural space. It may bulge slightly into the dural opening. Rupture of this membrane permits the pus to well up into the wound. When the pus has been evacuated, the compressed brain may once more expand into its original position beneath the dura. Its surface markings, however, may be obscured by the fibrinopurulent exudate still adherent to the outer surface of the arachnoid or by that within the subarachnoid space (associated leptomeningitis). The engorged, sometimes thrombosed veins may also be evident.

Characteristics of Concomitant Lesions.—As has already been intimated, subdural abscess of

Not always is there a large opening between the argin of the falx cerebri and the corpus callosum, e the arachnoid at times is adherent to that of the ite side in this region.

rhinogenic origin is often associated with a number of other lesions of the scalp, skull, dura mater, leptomeninges and brain, since one is concerned with a pathologic complex rather

group includes lesions both with and without skull—cellulitis of the scalp, epicranial abs focal or spreading osteomyelitis of the extradural abscess, thrombosis of the sup

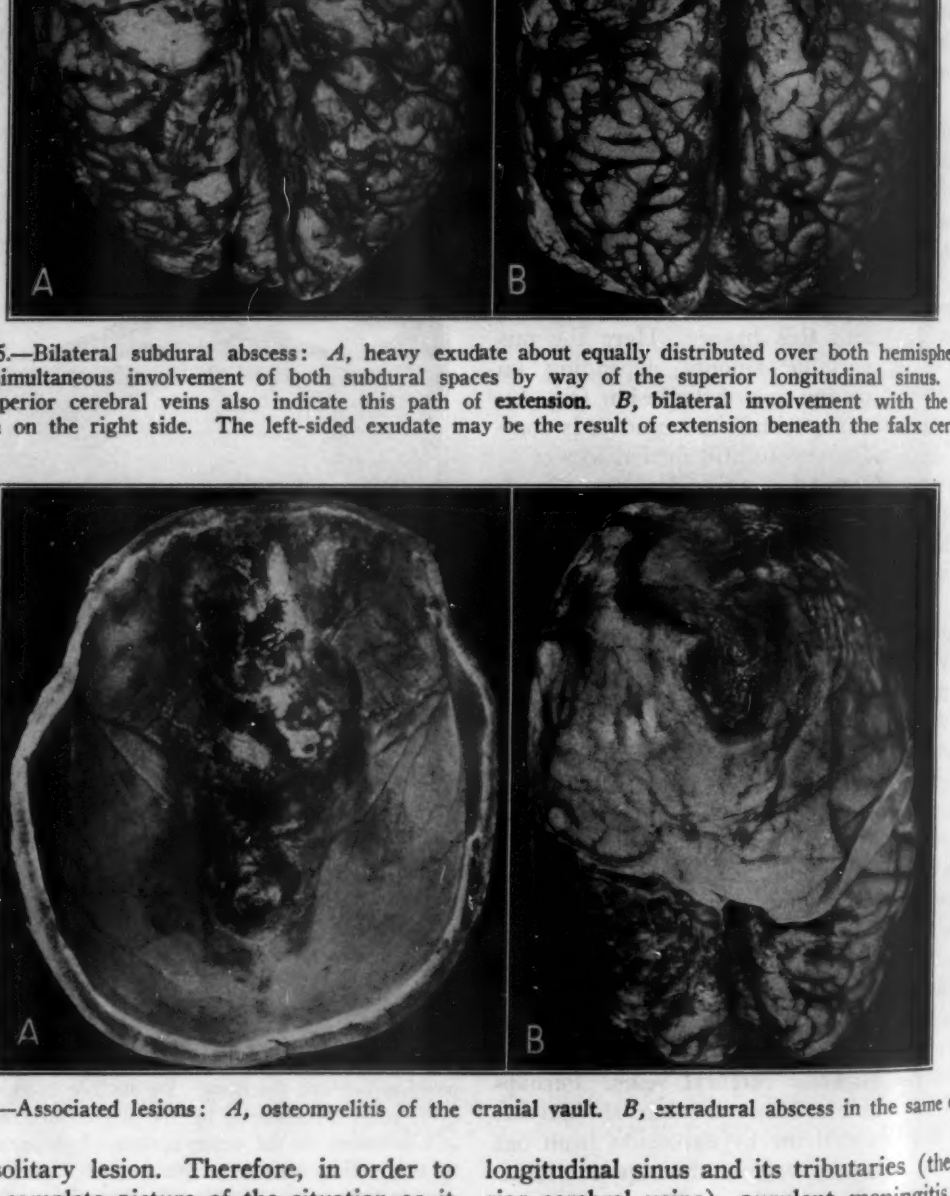


Fig. 5.—Bilateral subdural abscess: *A*, heavy exudate about equally distributed over both hemispheres gesting simultaneous involvement of both subdural spaces by way of the superior longitudinal sinus. Th bosed superior cerebral veins also indicate this path of extension. *B*, bilateral involvement with the gre extension on the right side. The left-sided exudate may be the result of extension beneath the falx cerebri

Fig. 6—Associated lesions: *A*, osteomyelitis of the cranial vault. *B*, extradural abscess in the same cas

than a solitary lesion. Therefore, in order to obtain a complete picture of the situation as it so often exists it will be necessary to include a description of those individual lesions with which subdural abscess is so often associated. The

longitudinal sinus and its tributaries (the s rior cerebral veins), purulent meningitis, c bral softening, focal encephalitis and absces the brain. Orbital abscess, which rarely occur these patients, is to be considered as a conco

development and not so much a link in the n of pathogenesis. It will therefore not be idered.

ellulitis of the scalp is usually located over affected frontal sinus or sinuses or over the re frontal region when associated with osteo- litis of the frontal bone. In the latter case east it is usually a question not of simple ma but of brawny induration resulting from infiltration of the tissues of the scalp. The face of the affected area appears bluish red, rm and warm to the touch and may pit on ssure (edema). The tissues of the scalp are ely vascular, bleeding profusely at the time urgical incision. Pus is not grossly evident in tissues in most cases. On cut section the olved tissues appear moist and highly vas- r.

picranial or subaponeurotic abscess is often ciated with cellulitis of the scalp and in fact be the more evident of the two lesions. It ost likely to be present when osteomyelitis he skull has developed. The lesion is some- s the result of a focal development around rombosed vein. It then begins as a local ess. It is not always easy to say whether subaponeurotic abscess (and cellulitis of the p) is a concomitant lesion, developing simul- eously from the infected frontal sinus, or ther it is instead a consequence of the osteo- litic process. When both cellulitis and sub- neurotic abscess are present and at all exten- , it seems more likely that they are the lt of this more widespread bony lesion.

Osteomyelitis of the skull, whether focal or eading, is not a common accompaniment of dural empyema. Conversely, in recognized s of osteomyelitis of the skull subdural in- ion is not particularly common. From these ervations one would conclude that these two ons result from essentially different routes extension from the frontal sinus. When both present, it seems likely that invasion has en place along both avenues more or less ultaneously. They are therefore concomitant ons rather than one being the necessary con- uence of the other. For this reason a detailed hologic description of osteomyelitis will not considered in this connection. It will be suffi- nt to say that the infection in the bone may a localized process involving the diploe and her one or both tables of the skull (localized ctious osteitis) or it may be a progressive cess extending by contiguity in the diploe more rapidly by way of the open and valveless loic veins (spreading osteomyelitis). The cted bone has a yellowish appearance and is soft in consistency. When the inner table is predominantly affected, a variable degree of extradural infection is also present (fig. 6). Pus may be grossly evident in the diploe. Ordinarily the localized process of infectious necrosis of the posterior wall of the frontal sinus, which may constitute an avenue of infection into the intracranial space, perhaps should not be considered as a distinct associated bony lesion.

Thrombosis of the superior longitudinal sinus and associated veins is not uncommonly present in cases of subdural abscess. This is a relatively frequent and significant lesion in the course of a study of the genesis of subdural empyema. In the great majority of cases the infection reaches the subdural space by way of the vascular channels. Even in those cases in which no gross venous lesion is found, in the absence of any other evident avenue the invasion is likely to have taken place by this route. The anterior portion of the superior longitudinal sinus is naturally most likely to be affected. The sinus may be occupied by a recent thrombus or by grossly evident pus (intrasinal abscess). In old lesions variable degrees of organization may be found. Such findings are rare, however, for spontaneous healing is uncommon.

Thrombosis of the afferent cerebral veins is a more common finding than is involvement of the sinus itself. In some instances at least this development is a result of the subdural infection, although in other cases it may well be the primary lesion. In either type of case these thrombosed vessels stand out as rounded firm blue-black cords, often marked along their course by whitish gray spots of thrombus clearly evident through their walls (fig. 5). These vessels may be partially obscured by the overlying exudate.

Leptomeningeal reactions may be local or generalized. While regional reaction of the soft meninges is almost invariably present at the time of death, generalized leptomeningitis is not often evident to any advanced degree. As suggested by study of the spinal fluid in the clinical stages of the disease, the arachnoid seems to be a remarkably efficient barrier to the spread of infection into the subarachnoid space. Often up to within a day or two of death the relatively few cells in the spinal fluid indicate that at most only a mild meningeal reaction has taken place. This is evidenced microscopically in sections of the meninges taken from the brain by an abundance of fibrin and a relative paucity of cells in the subarachnoid space subjacent to the fully developed subdural abscess. All stages between this picture and a fully developed, generalized

meningitis may be seen in any large series of cases. It should be repeated, however, that the patient usually succumbs as a result of the subdural abscess before the full-blown picture of suppurative meningitis develops either clinically or pathologically. The heavy deposit of either vertical or basilar subarachnoid exudate so commonly seen in cases of otitic meningitis is not often observed in cases of subdural empyema of frontal sinus origin.

In some instances septic meningitis may actually develop simultaneously with subdural abscess in those cases of infection via venous channels. This seems to be exceptional, however.

Red softening as a consequence of subdural empyema is usually the result of venous thrombosis and may be cortical and focal or subcortical and more widespread. There may be multiple foci of cortical softening, all fairly small. More extensive areas of red softening are more apt to be found in an infant or a young child. In addition to the grossly evident areas of softening, necrosis of the superficial or deeper layers of the cortex or of the entire cortex is often observed microscopically.

Abscesses secondary to rhinogenous subdural empyema fall into two general groups. The most common occurrence is that of one or more small or medium-sized, sharply limited but none too well encapsulated abscesses, which are usually subcortical in location. These stand out in contrast to the large abscess of the frontal lobe which occurs as an isolated complication of suppurative disease of the frontal sinus. [...] multiple, these smaller abscesses may be fo[...] both frontal lobes. The second group of ab[...] are found as a consequence of the more di[...] adhesive pachymeningitis interna; in ca[...] this type both intracerebral and isolated [...] dural abscesses are found. Two such case[...] included in the present series. Exampl[...] red softening and frontal abscess are show[...] figure 7.

Histologic Characteristics of Subdural Ab[...] —It is difficult to obtain an adequate conce[...] the microscopic characteristics of subd[...] empyema from blocks of tissue obtained [...] specimens brought to the laboratory, since by [...] time the brain and the dura have been remo[...] from the skull the lesion as such no longer ex[...] One has to be content with a study of a sect[...] from the dura mater whose inner surface is [...] coated with the typical varicolored exudate [...] with a section from the now isolated brain w[...] some of the exudate still adherent to its exter[...] surface. A mental reconstruction is still poss[...] by fusing the impressions gained from these t[...] sections from what was originally the t[...] boundaries of the accumulation of pus.

Sections through the dura mater disclos[...] layer of partially organized granulation tis[...] with free exudate still adherent to its in[...] surface. Unless one has by chance or des[...] taken the sections from an area of dura wh[...] the infection is passing through this membra[...]

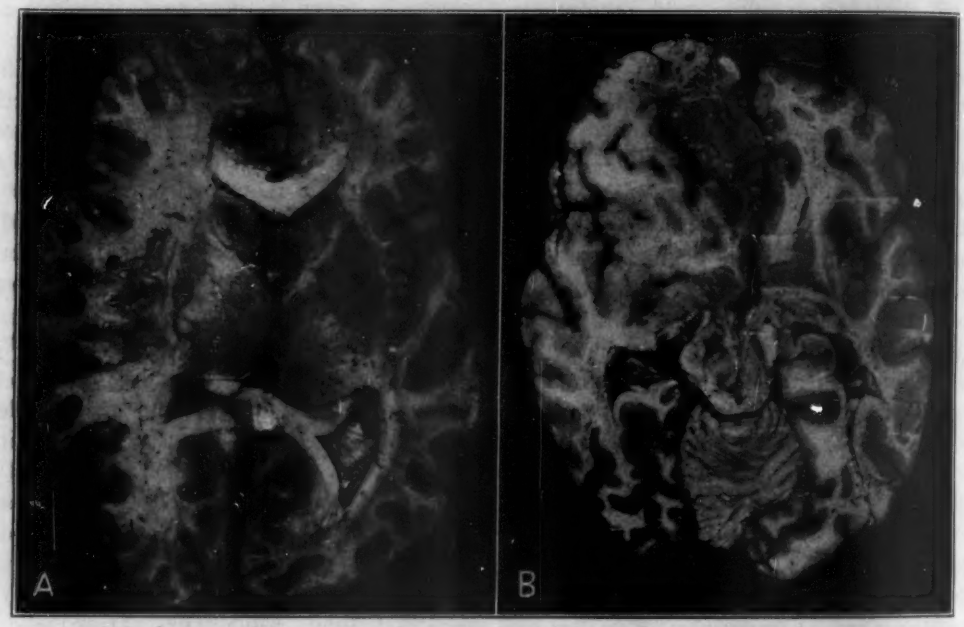
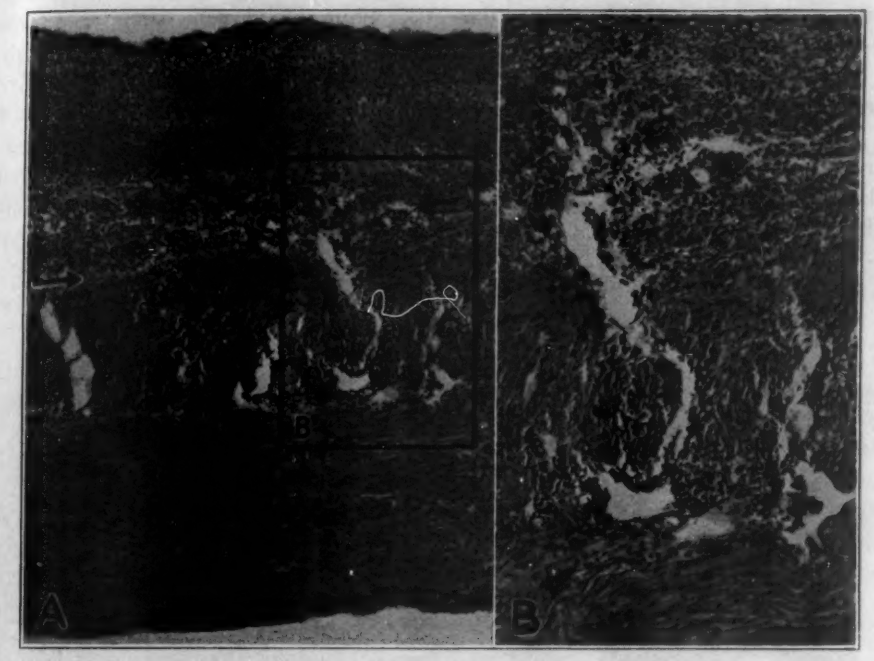
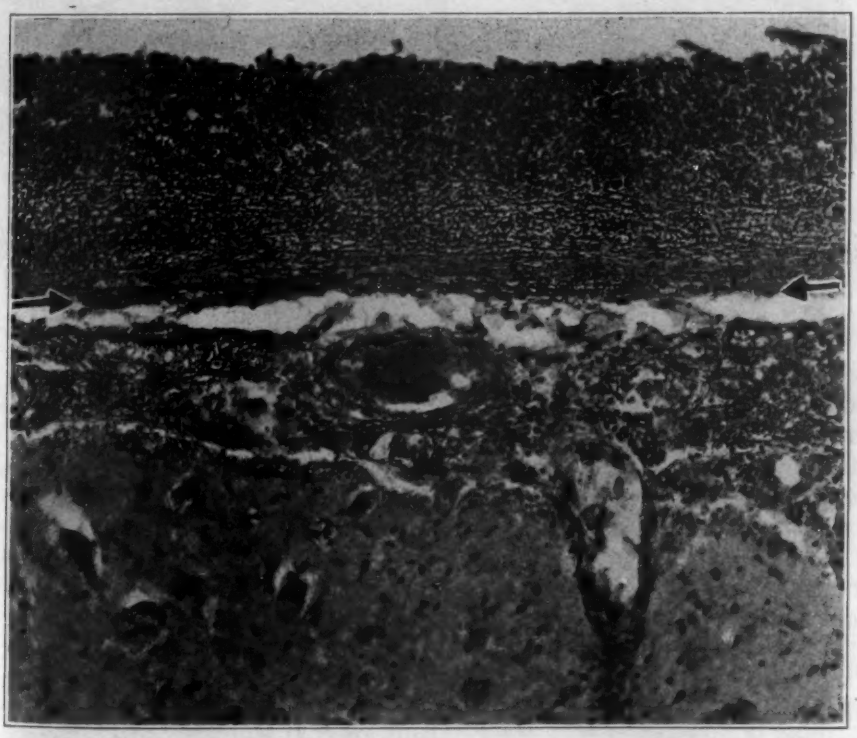


Fig. 7.—Associated lesions: *A*, foci of red softening in the cortex of the left frontal lobe. *B*, abscess of [...] frontal lobe.

an external suppurative focus (extradural s), the layer of granulation tissue is y sharply delineated from the inner surface dura to which it is adherent (fig. 8). In older lesions some evidence of invasion of the dura is sometimes found. At times the layer of new-formed mesothelial cells which constitutes the capsule of the abscess can be distinguished



g. 8.—Section through dura showing a layer of subdural exudate. *A* shows a fairly well defined margin en the exudate and the dura (arrows). *B* as inset shows leukocytes indicating invasion of infection about vessels deep in the inner leaf of the dura mater.



ig. 9.—Section through the leptomeninges and the brain. Arrows indicate the line of the arachnoid. The ate external to (above) the arachnoid is a part of a subdural collection of pus. The subarachnoid reaction yet largely fibrinous in character.

from the dura and its lining endothelium on one side and from the layer of granulation tissue on the other. As organization proceeds, however, this membrane becomes less apparent, the new connective tissue being quite intimately bound to the dura by the vascular sprouts penetrating the connective tissue layer from the dura. In time the adhesion becomes more and more organized until it is less easy to find the exact line of transition.

Sections through the cortex and the leptomeninges likewise present a layer of exudate external to the arachnoid (fig. 9). This layer is

another and in various regions in the same c
Early in the course of the lesion the spac
filled with fibrin in which various number
scattered leukocytes are found. Organisms
not present at this stage. As the lesion deve
the white blood cells become increasingly
quent until it is evident that localized menin
has resulted, as further evidenced by the a
presence of organisms in the exudate. Wi
progressive lesion, evidences of well develo
generalized meningitis may be found if
patient has survived for a sufficiently
interval.

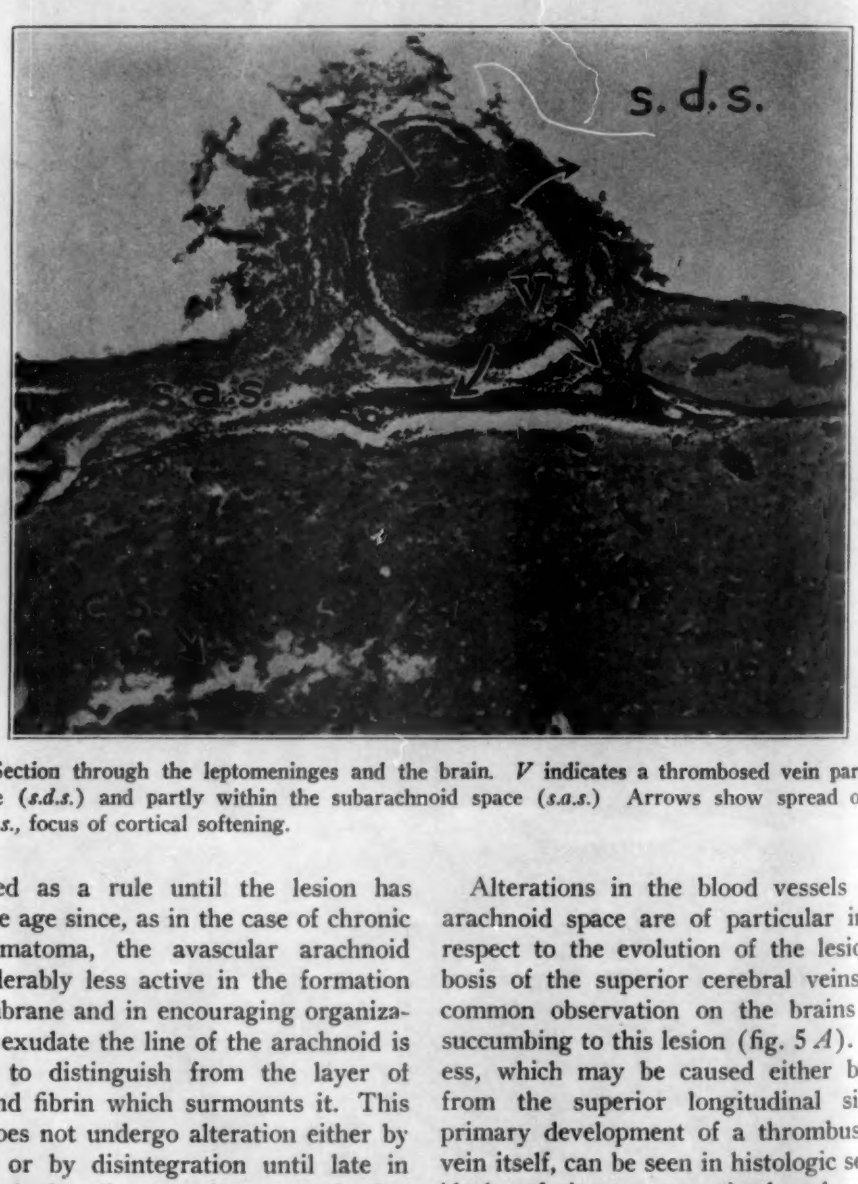


Fig. 10.—Section through the leptomeninges and the brain. *V* indicates a thrombosed vein partly within subdural space (*s.d.s.*) and partly within the subarachnoid space (*s.a.s.*) Arrows show spread of infection both spaces; *c.s.*, focus of cortical softening.

not organized as a rule until the lesion has attained some age since, as in the case of chronic subdural hematoma, the avascular arachnoid seems considerably less active in the formation of a neomembrane and in encouraging organization. In the exudate the line of the arachnoid is not difficult to distinguish from the layer of leukocytes and fibrin which surmounts it. This membrane does not undergo alteration either by proliferation or by disintegration until late in the course of the disease when organization ultimately binds the dura, the leptomeninges and the cortex into a solid scar.

The character of reaction in the subarachnoid space varies considerably from one case to

Alterations in the blood vessels in the s
arachnoid space are of particular interest
respect to the evolution of the lesion. Thro
bosis of the superior cerebral veins is a fa
common observation on the brains of pers
succumbing to this lesion (fig. 5 *A*). This pr
ess, which may be caused either by extens
from the superior longitudinal sinus or
primary development of a thrombus within
vein itself, can be seen in histologic sections fr
blocks of tissue appropriately taken. In fig
10 such a thrombosed vein can be seen in cr
section. It is obvious that this vessel wit
both the subdural and the subarachnoid space
suggestively disseminating infection in b

ows). This is made apparent by the exudate adherent to that portion of the vessel in the ural space and to the subjacent collection of in the subarachnoid space.

anges in the brain tissue itself may also be in these sections. Focal or diffuse softening or without ball or ring hemorrhages (de- ing on whether the softening is due to us or to arterial occlusion) is the usual ng. In some instances in which there is no sly evident change in the brain, histologic ons show considerable evidence of change, r focal or diffuse. Invasion of the cortex and subcortical white matter is not uncommon, focal abscess formation in all stages may be The walls of larger abscesses show variable ees of capsule formation, but this process rdinarily not far advanced in cases in which h is due to the presence of the subdural ess and its concomitant and consequent n.

istologic Characteristics of Healed Pachy- ingitis Interna.—In the relatively rare cases ealing, more or less complete organization s place, resulting in firm adhesions between dura mater, the arachnoid and the cerebral ex. In sections taken from the scar the ous layers may still be evident in the new- ed vascular connective tissue, in which col- ons of leukocytes are still present. In some ns localized abscesses in the subdural space still found with well organized granulation e linings. Leukocytes and occasionally nisms may be found in these circumscribed ns.

CLINICAL COURSE

survey of any considerable number of cases ubdural empyema secondary to suppurative se of the frontal sinuses makes it clear that complication when fully evolved presents a y typical clinical picture. Not only is this ut the predisposing situation also is rather estive. In this section will be considered the ntial infectious background which makes this n possible, as well as some details of its cal evolution and, finally, a description of ymptoms which may be produced by it.

fectious Background.—Since subdural empy- seems to be the direct result of an acute uration of the frontal sinus, it is of interest ote that the course of the disease is likewise e, with a survival period at times as short as w days. In the cases in which its evolution longer duration, acute infection of the frontal s is often found to be the cause of a second plication, such as osteomyelitis or thrombosis he superior longitudinal sinus, which, in turn, may have been the etiologic factor in the production of the subdural abscess. In some instances surgical drainage of the infected sinus has apparently delayed the development of the subdural complication. In this series it seems to be the exception to the rule when this lesion proves to be due to an exacerbation of a chronic sinus infection (4 of 42 cases). In the experience of other observers the opposite seems to be the case (8 of 12 cases).[28]

The clinical history of the original infection is stereotyped. Usually the sinusitis is introduced by a simple "cold" or the "flu." Less often but fully as significant is the development of an infection acquired at a community swimming pool. In some instances trauma to the frontal region either initiates or accentuates the sinus infection.

Symptoms and Signs.—The local infection is usually obvious. Pain, swelling and redness in the area over the affected frontal sinus (for in well over half the cases but one frontal sinus is involved) are conspicuous in this order of frequency. In an occasional instance the pain may be greater over the opposite frontal sinus, leading to mistaken lateralization of the sinus disease. Not infrequently the entire accessory nasal sinus system is infected (pansinusitis), in which case the symptoms are not so obviously focalized. Even in these circumstances, only one frontal sinus may be most specifically involved.

Regional complications often obscure the more lethal developments within the cranium. Local swelling of the scalp or of loose tissues of the eyelids or the evidence of more serious intra-orbital edema or abscess at times beclouds the picture. On occasion edema of the frontal region actually marks the development of focal or spreading osteomyelitis of the cranial vault, although ordinarily the presence of an infection of bone is indicative of a slower process whose duration may be measured by months or even years rather than days or weeks. The concomitant intracranial lesion in such instances usually proves to be pachymeningitis interna (often the cause of heavily encapsulated subdural abscesses and superficial cerebral abscesses) rather than acute subdural empyema.

The symptoms and signs of subdural empyema having its origin in disease of the frontal sinus may be divided into those indicative of a constitutional reaction to the subdural infection, evidences of increased intracranial pressure, signs of meningeal irritation and localizing symptoms and signs indicative of the seat of pressure. Not all of these groups of symptoms are elicited

in every case, because of the relatively small amount of exudate present in some instances and because some patients are not seen until in the terminal stage. The constitutional symptoms are usually the most conspicuous for obvious reasons, and meningeal signs are usually to be found if looked for. Phenomena indicative of increased pressure and localizing signs, dependent as they are on progressive accumulation of pus over the cerebral hemisphere, are not to be expected in those cases in which the exudate is limited in extent or amount.

Constitutional symptoms are invariably present in the form of fever and leukocytosis. The rise of temperature is variable in degree, ranging from 100 to 105 and averaging about 102 F. The temperature curve shows relatively small fluctuations in the average case, with a general tendency toward increasing elevation to the time of death. It is usually impossible to determine by this curve alone the exact time of invasion of the subdural space. Very likely the constitutional reaction to the sinus infection merges imperceptibly with that to the complicating subdural infection, which simply increases the area from which toxins can be absorbed. Chills have been complained of in less than a fourth of the cases of acute disease, although they were likely present to some degree in a greater percentage and the symptom would have been elicited if inquired for. Leukocytosis is quite obvious in all cases of acute disease and is present to some degree in the cases of chronic disease. The white blood cell count has varied between 12,700 and 37,500 per cubic millimeter, with an average count approximating 20,000 cells. The differential count is typically high in polymorphonuclear cells.

Symptoms and signs of increased intracranial pressure vary considerably from one case to another, both from a clinical and from a laboratory standpoint. Perhaps the most constant subjective symptom is headache, which is not always clearly evaluated by the patient or his examiners. It is not always evident from the history whether the headache is localized over the affected sinus or generalized because of increased intracranial pressure or is of the fronto-occipital type suggestive of meningeal irritation. Evidences of increased pressure manifested by changes in the optic disks have been recorded in about a fourth of the cases (11 of 42), but in many others such signs were obviously not looked for. As a rule, these signs were early and incomplete and were usually described as tortuosity and congestion of the optic veins, blurring of the disk margins and early elevation. The pulse rate is not always characteristic, although a relatively slow pul considered significant. Stupor, indicative of t increasing intracranial pressure and toxemi an almost invariable accompaniment, alw being present when pus has accumulated in great amount. Peculiarly enough, nausea vomiting as signs of increased pressure are ra mentioned. The actual degree of increase in p sure as measured by the spinal manometer been recorded as from 150 mm. (water pressu to over 400 mm. (the manometers in the hosp are 400 mm. long). Undoubtedly in many c the pressure was actually much greater. finer objective signs of increased engorgem of the veins of the scalp and the eyelids the elevation of the percussion note have been recorded if observed.

Signs of meningeal irritation in the form cervical rigidity, Kernig and Brudzinski sig and tache cérébrale are almost invariably pres before the cell count is elevated. Frank evide of purulent meningitis is not common except a terminal event.

Delirium rather than coma has been occasi ally recorded and suggests the occurrence cortical irritation rather than depression.

As has already been pointed out, it is occurrence of localizing or lateralizing sympto and signs which indicates the specific devel ment of a lesion affecting the cerebral he spheres. Such symptoms may be outspoken may be so minor as to be easily overlooked. is quite possible that in some cases of acu fulminating infections with which there is little exudate, these symptoms may be abs altogether.

The most conspicuous localizing sympto are motor in nature. Generalized convulsi suggest irritation of the motor cortex, but wi out some associated lateralizing phenomena th are not helpful in determining the side of lesion. Jacksonian seizures or local muscu twitching are important signs in this rega Such motor manifestations were described in of the 42 cases personally studied. Contralate motor weakness or paralysis, in whole or in pa was noted in 16 cases and abnormal cont lateral reflexes in 3 others. The paresis paralysis was described as spastic in some ca and flaccid in others. Among the 27 cases which the left frontal lobe (of the presum major hemisphere) was affected as the seat either a major or a minor subdural collection pus, motor aphasia was clearly evident or stron suggested in only 6 cases. The relative inf quency of this symptom is apparently due to fact that the exudate does not often accumul

ny great quantities this far down over the isphere.

ensory disturbances were rarely discovered, sibly because they were not more assiduously ght for or because the patient's mental state cluded careful examination. Hemihypesthesia described in but 2 of the 42 cases included in series.

ight homonymous hemianopsia was dis- ered in but 1 of the 4 cases of mesial abscess. ilatation of the pupil was the only sign sug- ing lateralization of the lesion in 2 cases, rring in both on the side opposite the sub- l abscess.

t may be said, therefore, that constitutional s are always present and usually form the st conspicuous manifestations of disease. Signs meningeal irritation become increasingly evi- t before the death of the patient. It is the easing prominence of evidences of increased acranial pressure and the occurrence of alizing signs which suggest the presence of umulated pus in one or the other subdural ce.

IRECT AND DIFFERENTIAL DIAGNOSIS OF SUBDURAL EMPYEMA SECONDARY TO FRONTAL SINUSITIS

he direct diagnosis of subdural abscess or pyema is not difficult in the majority of cases, e the possibility of the occurrence of the lesion appreciated and its relative frequency as an acranial complication is understood. The ure to diagnose this condition during life in great majority of cases is due to the fact t so few surgeons, either rhinologic or neuro- ic, understand the nature of the lesion. Occa- nally it is stumbled on in the course of an loration for a presumed rhinogenous abscess the frontal lobe.

n the usual case the clinical course is unmis- able. The development of isolated acute -sided frontal sinusitis (rarely an acute cerbation of chronic sinusitis) or of this dition as a part of pansinusitis with the usual al discharge in the course of an acute infec- n of the upper respiratory tract or influenza iates the disease. If sinusitis follows the use a community swimming pool or, as it occa- nally does, focal injury to the frontal region, it all the more suggestive. The infection of the ntal sinus usually is accompanied with local ns of inflammation—focal redness, swelling d tenderness—and sometimes with edema of eyelids. Almost imperceptibly the signs of al inflammation merge into signs of intra- nial extension, usually suggestive of menin- l irritation with frontal headaches, fever at times preceded by chills, and stiffness of the neck. It is only when symptoms or signs of a disturbance of cerebral function develop that the clinical observer is usually led to consider the diagnosis of an intracranial complication. These signs are usually of the nature of contralateral weakness or paralysis, either flaccid or spastic, convulsions, either typically jacksonian or generalized with lateralizing phenomena, and motor aphasia if the major hemisphere is compressed. Hemihypesthesia and homonymous hemianopsia (mesial subdural abscess) are much less commonly elicited. These lateralizing symptoms and signs are not always clearly and unmistakably present. Sometimes only a minor difference in the deep and superficial reflexes and the presence of pathologic reflexes constitute the sole evidence of a subdural abscess, signs that may never be discovered unless carefully searched for.

One sign which helps in distinguishing a subdural from an intracerebral abscess is a relatively slow pulse in the presence of a moderately elevated temperature.

Signs of meningeal irritation are often present, particularly in the terminal stage, but signs of full-blown purulent meningitis are usually wanting even just before death. Increased spinal fluid pressure with minor to moderate pleocytosis in a clear or opalescent fluid without organisms on smear or culture is the rule.

There are some cases in which it is extremely difficult or impossible to make a correct diagnosis. At times the diagnosis of the original frontal sinusitis is not definitely established, which would mislead the observer to overlook this condition as the possible cause of an obvious intracranial lesion. In other cases the minimal lateralizing signs are overlooked, and the nature of the intracranial complication is not appreciated.

TREATMENT

The treatment of subdural abscess secondary to frontal sinusitis is primarily and basically surgical. While in some instances adequate administration of the appropriate sulfonamide compounds may prevent the development of subdural abscess, an acute purulent empyema of the frontal sinus should nevertheless be considered a surgical lesion. Reasonably early evacuation of the pent-up pus by external trepanation of the sinus will very likely be sufficient to prevent the development of intracranial complications. The intranasal approach to such a lesion seems to be distinctly outmoded, about as productive of harm as of benefit. The very early intracranial extension to the subdural space from an empyema of the frontal sinus in many cases seems to make idle any talk of delay in evacuation of the sinus

in the vain hope that nature will establish an effective barrier to such extension. The infection undoubtedly spreads very early by way of regional vessels not yet occluded by the products of inflammation.

It is likewise futile to hope that the sulfonamide compounds can cure such a lesion once it is definitely established. Their administration may even serve to obscure the picture by lessening the constitutional and localizing phenomena and may actually reduce the amount of, and change the character of, the inflammatory exudate. The ineffectiveness of this form of therapy lies in the fact that the subdural space has a poor arterial blood supply and little concentration of the drug in the heavy exudate in this space is to be expected.

The only hope of cure is in surgical drainage of the abscess through one or more trephine openings over the cranial vault with placement of tubular soft rubber drains well distributed in the subdural space. It is probably advisable when one is uncertain to make openings on both sides. The use of sulfonamide drugs after drainage is advisable both locally and systemically. The dried crystals should be blown into the exposed and emptied subdural space at the time of operation.

Because appropriate surgical measures have not been applied in any great series of cases the prognosis with this form of treatment is yet problematic.

SUMMARY AND CONCLUSIONS

This study is concerned with the most common yet least understood intracranial complication of suppurative frontal sinusitis—subdural abscess or empyema. This lesion, which i well defined pathologic entity, provokes a fa typical clinical syndrome when the pus accu lates in sufficient quantities over the cere hemisphere. Usually the result of acute sup rative frontal sinusitis ("empyema"), o limited to but one side, subdural abscess u commonly develops from a blood-borne in tion which reaches the subdural space via superior longitudinal sinus and its venous co munications.

As in the case of its precursor lesion, subdu abscess is usually an acute and rapidly progr sive lesion. In the great majority of cases i lethal. The pus when unobstructed accumul rapidly and spreads with little interference the subdural space, giving rise to contralat motor symptoms and signs associated with m aphasia if the major hemisphere chances to the one affected. When the infection is fulminating, pachymeningitis interna results w ultimate fusion of the dura, arachnoid, pia a cerebral cortex and the consequent formation localized, heavily encapsulated subdural a intracerebral abscesses which pursue a prolon and heart-breaking clinical course.

When it is realized that subdural empye with origin in the frontal sinus is about fi times as frequent as the better known and m commonly suspected abscess of the frontal lo the occurrence of one-sided motor phenome will more often lead to trepanation of the sk and exploration of the subdural space, and su lives will be saved which otherwise will inevita and certainly be lost. Undrained surgically t lesion always proves fatal to its unluc possessor.

401 Bailey Street.

ADENOMA OF THE TRACHEA AND THE BRONCHUS

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he belief existed until recent times that the s and bronchi were practically immune to ary neoplasms. Statistics disclose that there tually a predilection of primary tumors for e organs and that 12 to 15 per cent of all ers originate in them.

nother erroneous belief was that all primary bronchial tumors were malignant. In the of more recent knowledge one finds that 10 per cent of such tumors are benign. majority of the benign tumors have suf- nt common histologic and clinical char- ristics to constitute a distinct entity. This oday designated "adenoma." Adenoma is e distinctly segregated from carcinoma which closely resembles histologically. Because his close resemblance tumors of this group years were classed as malignant; it remained clinicians who followed the course of patients letermine that they were benign. "Benign" his instance is not synonymous with "harm- " nor does it imply that the growth is in- able of terminating the life of the afflicted son.

SYNONYMS

he term "adenoma" embraces a large group umors which have been described as benign noma, polypoid adenoma, adenomatous polyp, noma of the bronchial mucous glands, carci- d, benign epithelial tumor, benign glandular or, adenocarcinoma of low grade malignancy, mor-like formation of inflammatory hyper- ia," endothelioma, mixed tumor, and so on.

TOLOGIC ORIGIN OF PRIMARY ENDOBRONCHIAL TUMORS

he primary bronchi are lined by pseudo- tified ciliated columnar epithelium. The hea is lined by true ciliated columnar epi- ium. Many tubuloacinous mucous glands present in the submucosa of both the trachea d the bronchi. The more frequently encoun- ed squamous cell carcinoma originates in the al cells of the mucosal epithelium. The less quent adenocarcinoma and adenoma originate m the acinous cells or duct cells of the sub- cosal tubuloacinous mucous glands.

Many of the tumors classed as adenoma show tinct alveolar or acinous arrangement, sug- ting origin from gland cells, while others show strandlike arrangement of cells in parallel cylindromatous formations highly suggestive of origin from gland ducts.

Adenoma, originating as it does in the submucosa, lifts the mucosa over its surface as it grows; therefore the mucosa presents itself to the endoscopist's eye as the smooth glistening surface of the tumor.

As the tumor grows and the lumen of the trachea or the bronchus becomes partially or totally filled with the mass, molding and encasing pressure are exerted on it by the rigid bronchial or tracheal cartilaginous frame, imparting to the tumor a rounded and elongated form. The extrusive force of the cough causes the free terminal end of the mass to present upward toward the mouth, a fact that has frequently been noted endoscopically and at postmortem examination.

A study of the rapidly accumulating literature on the subject has caused me to wonder whether this growth in the female might not be distinct from that occurring in the male and, in fact, arise from primordial gonadal cell rests. The recurring hemoptyses that frequently are concurrent with the menses, as well as the fact that these patients frequently give a history of pulmonary bouts beginning at the menarchal period, seem to give support to this conjecture. These attacks are usually diagnosed at the time as pneumonia, pneumonitis, paroxysms of coughing, hemoptysis and so on; they frequently recur with the menses. They are, of course, due to the undetected presence of adenoma in the trachea or the bronchus.

TUMOR GROWTH AND EXTENSION

Adenoma may be pedunculated or may have a sessile base. The tumor with the thin pedicle, as a rule, grows entirely within the bronchial lumen. The intramural type presents a flat base and grows between the cartilaginous rings, elevating the bronchial mucosa inwardly and the fibrous membrane outwardly but remaining encapsulated within this limiting membrane. The endoextrabronchial type has the configuration of a dumbbell and for this reason has been called the dumbbell type. The constriction takes place at the bronchial wall. The smaller portion presents endobronchially and the larger extra-

bronchially. According to Brunn and Goldman,[1] only the pedunculated, entirely endobronchial growth is amenable to complete cure through bronchoscopic means. The others demand adequate surgical intervention since their extramural extensions encroach on surrounding lung tissue and are impossible to remove successfully by other than surgical means.

SYMPTOMS AND PHYSICAL SIGNS

Adenoma presents no findings which are peculiar to itself. The findings are those characteristic of bronchial obstruction and depend on the location of the tumor, the type of obstruction produced, the amount of tributary alveolar tissue obstructed and other factors, such as the type and the virulence of secondary infection.

Cough and profuse hemoptysis are the two most frequent and constant signs of adenoma.

Fig. 1 (case 1).—*A*, roentgenogram of the chest before removal of an adenoma from the left main bronc showing atelectasis of the left lung with displacement of the cardiac and mediastinal shadows to the left. incorrect roentgen diagnosis of consolidation and pleural effusion was made.) *B*, roentgenogram taken a removal of the adenoma. There is almost complete reexpansion of the left lung. The patient is entirely symp free, and there has been no recurrence of the tumor. There remains, however, some atelectasis of the base of left lung.

Biermer,[2] in 1860, described the morbid processes that developed in lung tissue distal to a point of obstruction. The healthy pulmonary tract presupposes constant patency and adequate aeration. When obstruction takes place, a favorable medium is provided for anaerobes, saprophytes and other bacteria. The resulting sequelae are pneumonitis, bronchiectasis, abscess, fistula, empyema and other suppurative and necrotizing lesions.

Chevalier Jackson,[3] in 1930, published his classic "The Mechanism of Physical Signs in Neoplastic and Other Diseases of the Lung." In this work he described the three types of br chial obstruction, comparable in action to three types of valves in mechanics, produced one or more of the following conditions: narrowing or enlargement of the lumen; swelling of the mucosa; (3) projecting of abn mal tissue; (4) presence of a foreign body o secretions acting as such (endogenous for body). The valvelike obstructions were h to be as follows:

1. Stop valve obstruction: The bronchus is c pletely occluded or corked, and air cannot be insp or expired.

2. By-pass valve obstruction: There is partial struction or narrowing of the lumen. Air can pas and out of the lung but only in limited amounts. asthmatoid wheeze, heard at the open mouth, is ch acteristic of this mechanism.

3. Check valve obstruction: The occluding body terferes with either the inspiratory or the expira phase of respiration leaving the other phase unimpai either emphysema or obstructive atelectasis results, pending on the setting of the check valve.

Adenoma is capable of reproducing any one these phenomena. Since the growth obstru the major bronchi, a consideration of the roe genologic manifestations is given.

ROENTGENOLOGIC MANIFESTATIONS OF BRONCHIAL OBSTRUCTIONS

Obstructive atelectasis is manifested by di nution in the alveolar capacity on the involv side of the thorax and elevation of the diaphra on the same side. The ribs are arranged in plane that is more vertical than the normal, can readily be seen by comparison with

1. Brunn, H., and Goldman, A.: The Differentiation of Benign from Malignant Polypoid Bronchial Tumors, Surg., Gynec. & Obst. **71**:702-722, 1940.

2. Biermer: Zur Theorie und Anatomie der Bronchienerweiterung, Virchows Arch. f. path. Anat. **19**:94, 1860.

3. Jackson, C.: The Mechanism of Physical S in Neoplastic and Other Diseases of the Lung, J M. A. **95**:639 (Aug. 30) 1930.

site or unaffected side. The intercostal es are narrowed. The tracheal, mediastinal cardiac shadows are displaced toward the ctatic side. Atelectasis of the right lung lts in displacement of the right border of the t to a point well within the right thoracic y and of the apical or left border toward atelectatic lung often appears clouded as a result of pneumonitis or "drowned lung."

Obstructive emphysema is manifested by displacement of the heart and the mediastinum toward the contralateral side or the side opposite to the obstruction. When obstructive emphysema is suspected, it is mandatory that roentgen-

Fig. 2 (case 1).—Photomicrograph (×46). Note the prominence of vascular spaces and the compact collec- of cells surrounding these spaces. United States Army Medical Museum negative no. 67972.

midline. Atelectasis of the left lung displaces entire cardiac shadow extremely to the left; the posterior-anterior roentgen plate of the t the right border of the vertebral column istinctly discernible. Ordinarily this is not owing to the dense cardiac shadow superosed over the lower thoracic vertebrae. The ograms be taken both on full inspiration and on full expiration. The roentgenogram taken on full inspiration will show full and equal expansion of the two sides. On full expiration, however, the abnormally ballooned obstructed lung fails to contract as does the normal unobstructed lung. The unaffected side shows contraction on expira-

tion and expansion on inspiration, while the obstructed side remains ballooned on both. The roentgen shadows of the heart, the trachea and the mediastinum are normally placed on inspiration; on expiration, however, these organs are forcibly pushed by the overexpanded lung into the opposite cavity. The intercostal spaces on the emphysematous side are widened as compared with those on the normal side, and the ribs assume a plane more horizontal than the normal.

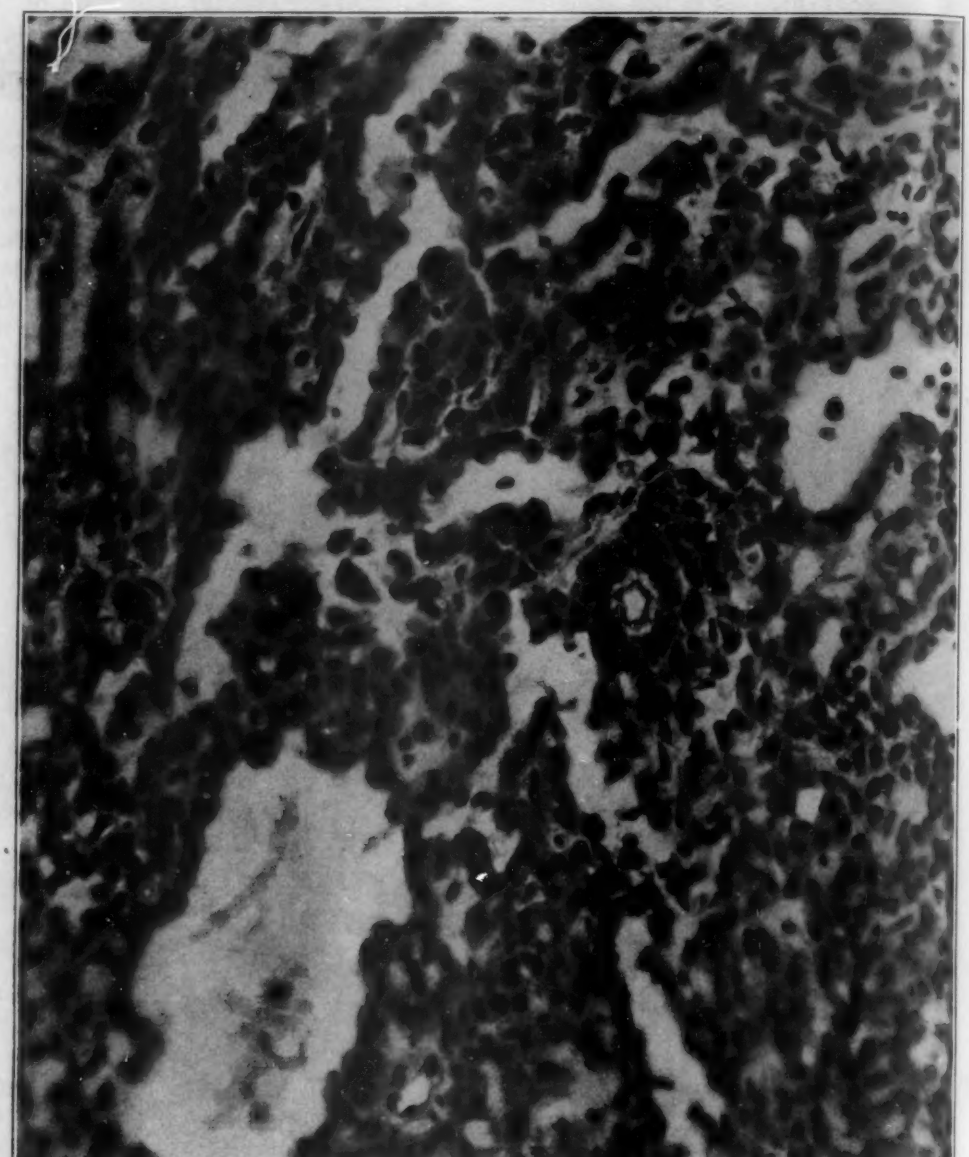

Fig. 3 (case 1).—Photomicrograph (×441) showing the character of the tumor. The cells are arranged in anastomosing strands. While the cells are uniform in size, great variation in shape from spindle to oval to round is present. Note the tendency to acinous arrangement. United States Army Medical Museum negative no. 67945.

DIFFERENTIAL DIAGNOSIS

The lesion with which bronchial adenoma is most frequently confused is bronchogenic carcinoma. Both produce bronchial obstruction, and the same sequelae of obstruction are met with in both conditions.

Adenoma is overwhelmingly a disease of young women. Of 12 cases reported by Jackson and Konzelmann,[4] 11 occurred in women. The diagnosis of the lone exception was inconclusive, pathologic sections having been lost many years before; the diagnosis in this case, therefore, was purely clinical and was without histologic corroboration.

4. Jackson, C. L., and Konzelmann, F. W.: Bronchoscopic Aspects of Bronchial Tumors, J. Thoracic Surg. **6**:312, 1937.

ost cases of adenoma in women occur
een the menarche and the menopause, or
g the years of fertility.
rcinoma, on the other hand, is preponder-
a disease of old men. The ratio of male to
le patients is between 2 and 3 to 1.
here is some evidence that adenoma at times
rgoes cancerous transition. Wessler and
gested the possibility of a similar transition in this case.

Bronchoscopically, carcinoma gives the appearance of a smooth, glistening, somewhat translucent and often lobulated tumor. Frequently it is pedunculated and is encased in a nonfixed or nonrigid bronchus. Carcinoma of the bronchus frequently has a fungating, ulcerated and papil-

Fig. 4 (case 1).—Photomicrograph (× 426) showing tumor bulging into a blood vessel. Note the palisading ells. United States Army Medical Museum negative no. 67944.

in[5] reported 2 cases in which such transmation took place. Chevalier Jackson reported ase of adenocarcinoma of the breast in a man who had previously been treated for nchial adenoma on the same side. He sug-

lary appearance. The bronchus, as a rule, is partially or totally stenosed and is rigid or "frozen" to the touch.

Hemoptyses occurring with adenoma are inclined to be much more profuse than those with carcinoma and are frequently encountered during the menstrual period.

5. Wessler, H., and Rabin, C. B.: Benign Tumors the Bronchus, Am. J. M. Sc. **183**:164, 1932.

Adenoma does not metastasize nor does it infiltrate surrounding tissue; it does, however, recur and encroach on the surrounding lung tissue. Adenoma remains encapsulated.

While carcinoma kills by virtue of those properties common to all cancers, adenoma if inadequately treated destroys life by obstructing the bronchus and thereby setting in motion dangerous and fatal complications.

Any condition which produces bronchial obstruction might be confused with adenoma. Frequently bronchoscopy alone can produce the proper diagnosis and is practically always necessary if a proper diagnosis is to be made.

TREATMENT

Until the present time bronchoscopic treatment, consisting of gross removal of the tumor with biting forceps followed by electrocoagulation of the base through the bronchoscope, has been deemed adequate for effecting a complete cure of adenoma. Frequent recurrences after apparent cures have demonstrated fully the fallacy of this assumption. Resort to radium and roentgen rays has proved entirely ineffectual in both

Fig. 5 (case 2).—Low power photomicrograph (×107) of bronchial adenoma. United States Army Medical Museum negative no. 68586.

lication and the retardation of growth of e tumors.

seems likely at this time that the tumors arise from stalklike bases and have neither al nor extrabronchial extensions can be rely cured by bronchoscopic means. This

It is now generally conceded that those tumors which show mural and extramural extension and all cases complicated with irreparable pulmonary damage as a result of prolonged obstruction demand surgical intervention. Eradication of diseased pulmonary tissue must be thorough.

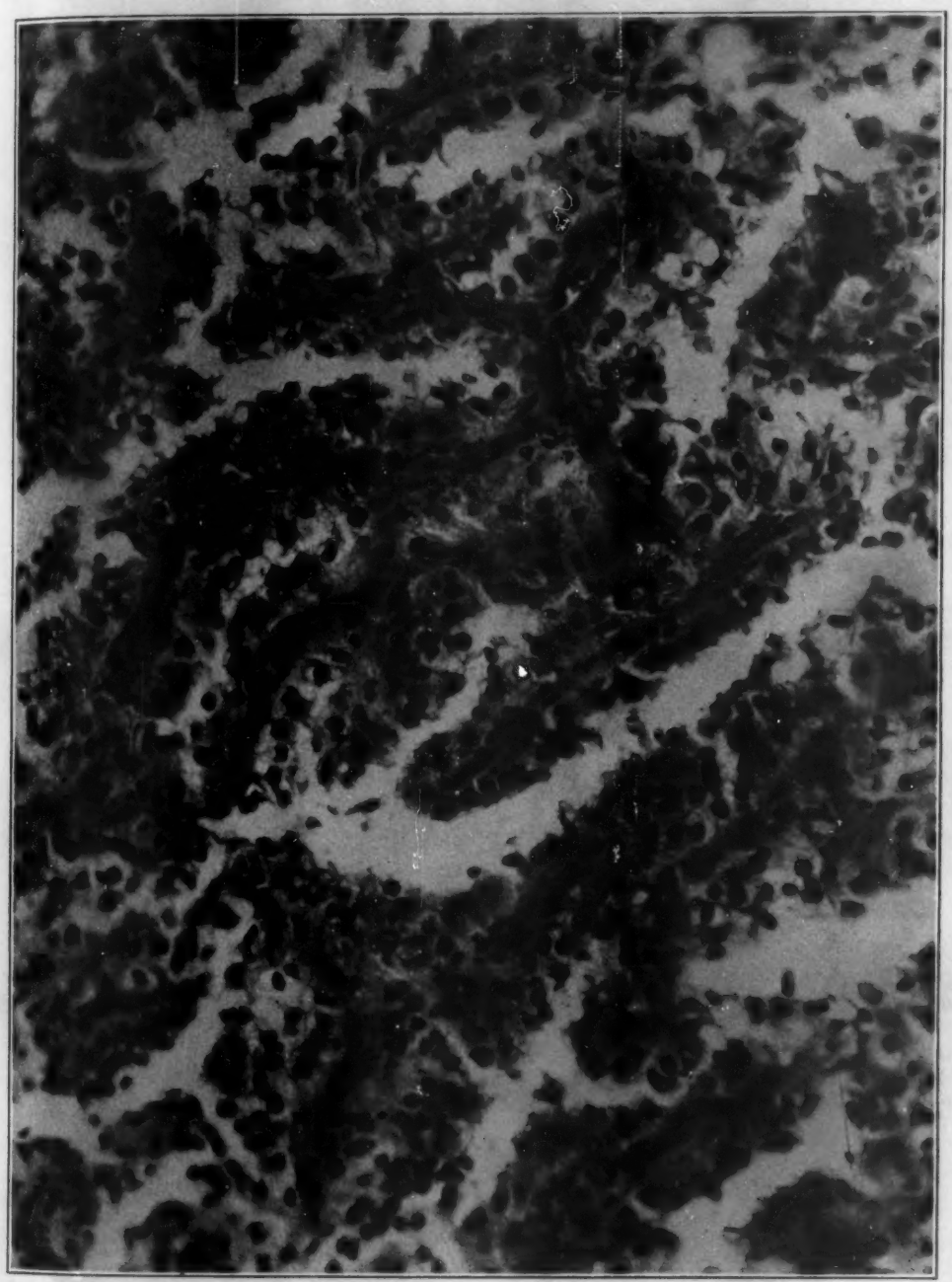

Fig. 6 (case 2).—High power photomicrograph (× 444) of adenoma of the bronchus. United States Army dical Museum negative no. 68586.

plies to the growth alone and not to patho- ic states of the lung distal to the growth which ght demand more drastic treatment. In those es which are uncomplicated, therefore, bron- scopic management might suffice.

REPORT OF CASES

CASE 1.—Mrs. H. W., a 44 year old white housewife who was born in Austria, was admitted to Trinity Hospital, Brooklyn, on Oct. 10, 1937. Her chief complaint was "warmth and chills; restlessness; cough productive

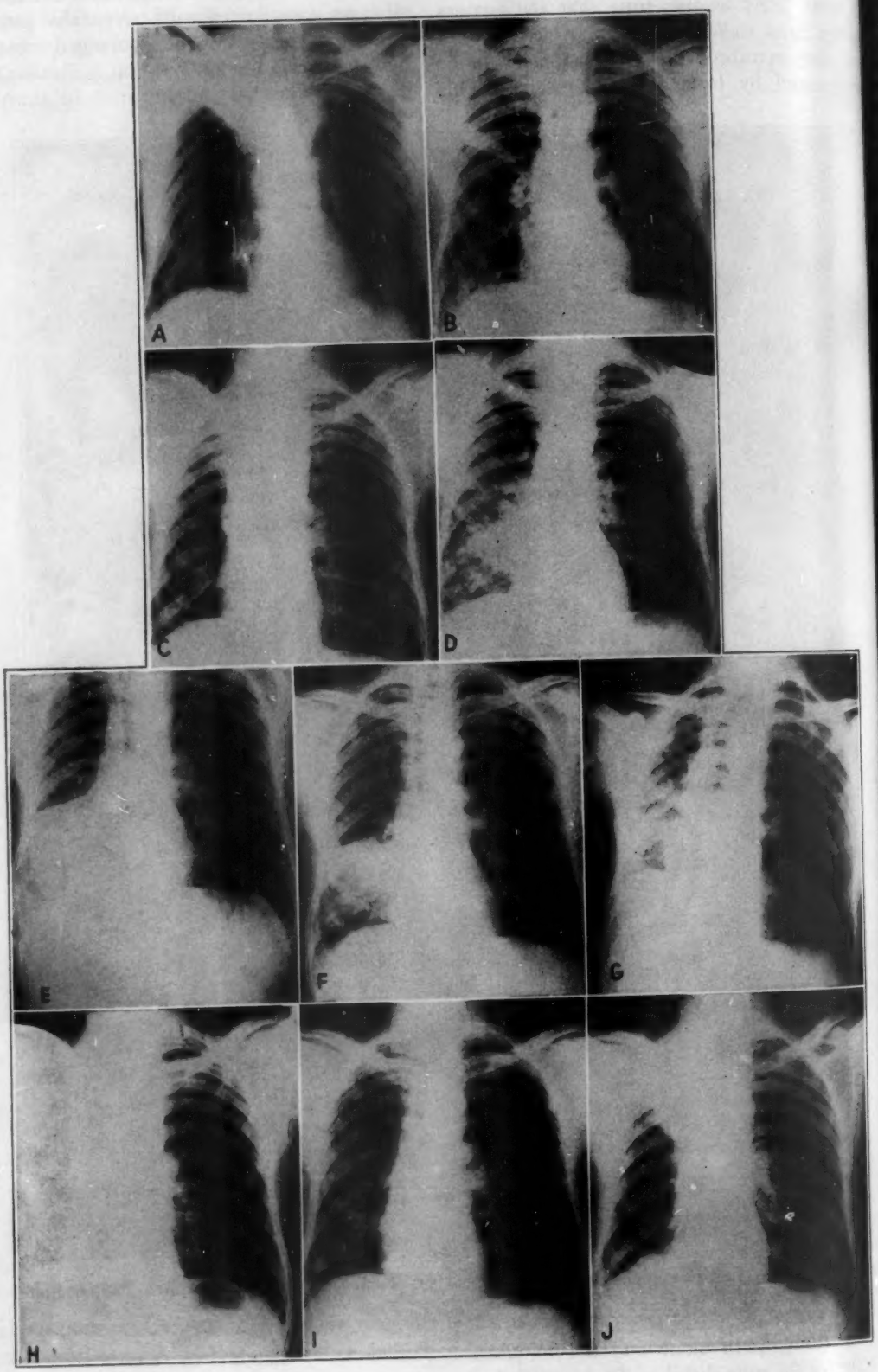

Figure 7

(See legend on opposite page)

white, and on one occasion rusty, sputum." The gh had persisted for one year, and her condition had n diagnosed as bronchitis. Bronchograms with in- ion of iodized poppyseed oil 40 per cent had been en twice during the year. Many examinations of um and fluoroscopic and roentgenologic examina- s were also made at Kings County Hospital, the lts of which were not disclosed to the patient. She attended the clinic for diseases of the chest at the w York Post-Graduate Medical School and Hospital. stated that on her last visit there she had expec- ted small bits of blood on several occasions over a iod of one week. A roentgenogram taken on April 21, 7 was reported as showing only "a slight degree of hilus and root branch thickening."

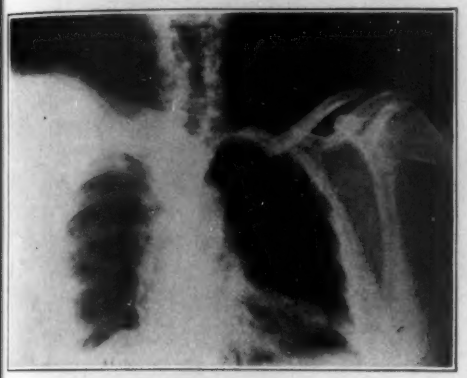

Fig. 8 (case 3).—Roentgenogram showing adenoma the trachea with the angulation and displacement.

On admission to Trinity Hospital she had been con- ed to bed for the previous eight days, and because re was no improvement she desired hospitalization. The doctor admitting her noted that she was cyanotic d dyspneic and appeared acutely ill. Respirations re labored, and there was an expiratory grunt. There s dilation of the alae nasi.

The clinical diagnosis was pneumonia of the lower lobe of the left lung whereas the roentgenologic opinion was (1) pleural effusion and (2) consolidation.

When the findings persisted for six weeks bronchoscopic examination was requested (fig. 1A).

The presence of adenoma was suspected because of the obstructive atelectasis, which was evident from the roentgen film, and the hemoptyses in an otherwise robust young woman. The suspicion was corroborated by bronchoscopic examination on November 24. Displacement of the trachea, the carina and the left main bronchus to the left was found. After a large amount of mucopurulent discharge had been aspirated from the left main bronchus, a large, smooth, round, multilobulated and semitranslucent mass was found completely occluding this bronchus. It was found attached to the upper lip of the bronchial branch supplying the superoposterior segment of the lower lobe of the left lung sometimes referred to as the apical branch of this lobe.

After the tumor had been removed, the roentgenogram showed reexpansion of the involved lower lobe with slight residual atelectasis (fig. 1 *B*). This has persisted.

One week later the base of the tumor was destroyed with monopolar electrocoagulation, the active electrode having been inserted through the bronchoscope. When the patient was examined bronchoscopically in January 1939, no recurrence was found, and a recent telephone conversation with the patient revealed that she had no further difficulty.

The pathologists consulted diagnosed the tumor as follows (figs. 2, 3 and 4): Dr. Max Lederer: "New growth resembling perithelioma." Dr. James Ewing: "Adenocarcinoma of the type described by Clerf—as running a rather benign course and often failing to recur after local removal." Dr. Shields Warren: "Benign adenoma." Col. J. E. Ash, curator of the Army Medical Museum: "Benign adenoma."

This case was described in the Otolaryngic Atlas published by the Army Medical Museum.

Case 2.—H. S., a 64 year old white man, was admitted to the Beth Israel Hospital, New York, April 7, 1939. His chief complaint was cough and expectoration

Explanation of Figure 7

Case 2.—Serial roentgenograms showing the changes accompanying the intermittent aeration and obstruction ulting from bronchoscopic removal of obstructing tumor tissue and regrowth of the tumor.

A—April 7, 1939: Appearance on admission to the hospital. Atelectasis of the upper lobe of the right lung seen.

B—April 10, 1939: Reexpansion of the upper lobe of the right lung following bronchoscopic removal of tissue structing this bronchus. A small area at the base of the upper lobe remains atelectatic.

C—May 12, 1939: One month later there is a recurrence of the atelectasis of the upper lobe of the right g with displacement of the mediastinum to the right of the midline. Inflammatory changes are noted at the se of the lung. It is evident that regrowth of the tumor again has caused obstruction of the bronchus.

D—June 19, 1939: About one month later reexpansion of the upper lobe of the right lung and return of mediastinum to the midline have followed bronchoscopic removal of the obstructing tissue. Marked pneumonitis the lower two thirds of the lung has developed as a result of prolonged obstruction.

E—June 24, 1939: Five days later atelectasis of the middle lobe and probably of the lower lobe of the right g has appeared with displacement of the mediastinum to the right. The upper lobe of the right lung is well rated at this time.

F—June 27, 1939: Three days later there is partial regression of the atelectasis. The mediastinal shift is ll present.

G—July 1, 1939: Four days later there is recurrence of atelectasis of the upper lobe with marked mediastinal splacement.

H—July 14, 1939: There is atelectasis of the entire lung with the mediastinum retracted into the right mithorax.

I—Aug. 16, 1939: Following several bronchoscopic treatments for removal of obstructing tumor, the lung s reexpanded, although the mediastinum remains slightly displaced to the right. A dislocated radon seed can be seen.

J—Jan. 4, 1940: Atelectasis or consolidation of the upper lobe of the right lung is present and pleurodiaragmatic adhesions. Note the similarity to *A* after the lapse of almost a year.

of bright red blood; he felt feverish and weak. His illness began five days before admission, with chills and fever, hemoptysis and coughing which were so severe that vomiting followed. He stated that he had noted the onset of the cough and hemoptysis in November 1938, six months before. Roentgen examination showed obstructive atelectasis. Carcinoma was suspected, but in view of the severity of the hemoptyses Dr. Ha Wessler favored the diagnosis of adenoma.

On bronchoscopic examination a tumor was found the upper portion of the main bronchus, occupying large portion of the lumen. It arose from the latera wall of the bronchus at the lower lip of the upp orifice. All visible growth was removed. The patho

Fig. 9 (case 3).—Photomicrograph (×21) showing the general topography of about two thirds of the lesi United States Army Medical Museum negative no. 72510.

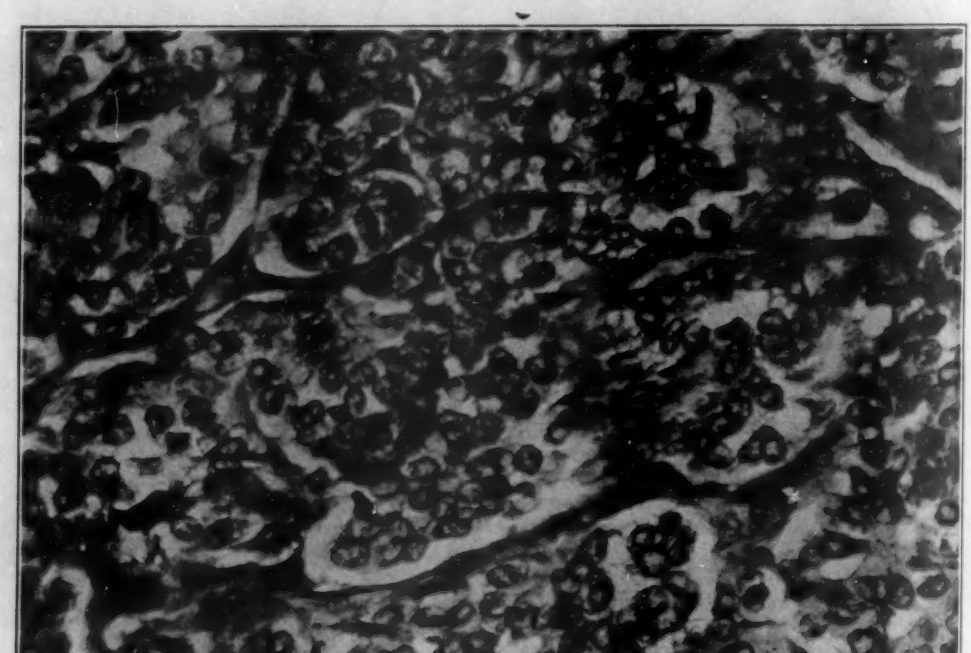

Fig. 10 (case 3).—Alveolar "base" of tumor (×480). United States Army Medical Museum negati no. 72511.

ic diagnoses were (figs. 5 and 6): Dr. Alfred Plaut: denoma." Col. J. E. Ash: "Adenoma of doubtful lignancy."

Treatment consisted of fifteen endoscopic procedures thin the following seven months. At each operation available growth was removed to reestablish ventiion of the lung. Electrocoagulation was repeatedly died some time later of a cerebral accident. Permission for an autopsy was not obtained.

It is noteworthy that while the proper microscopic diagnosis was adenoma, the clinical course of the disease in this instance was entirely different from that in the other 2 cases reported. The tumor was tough and firm and recurred rapidly.

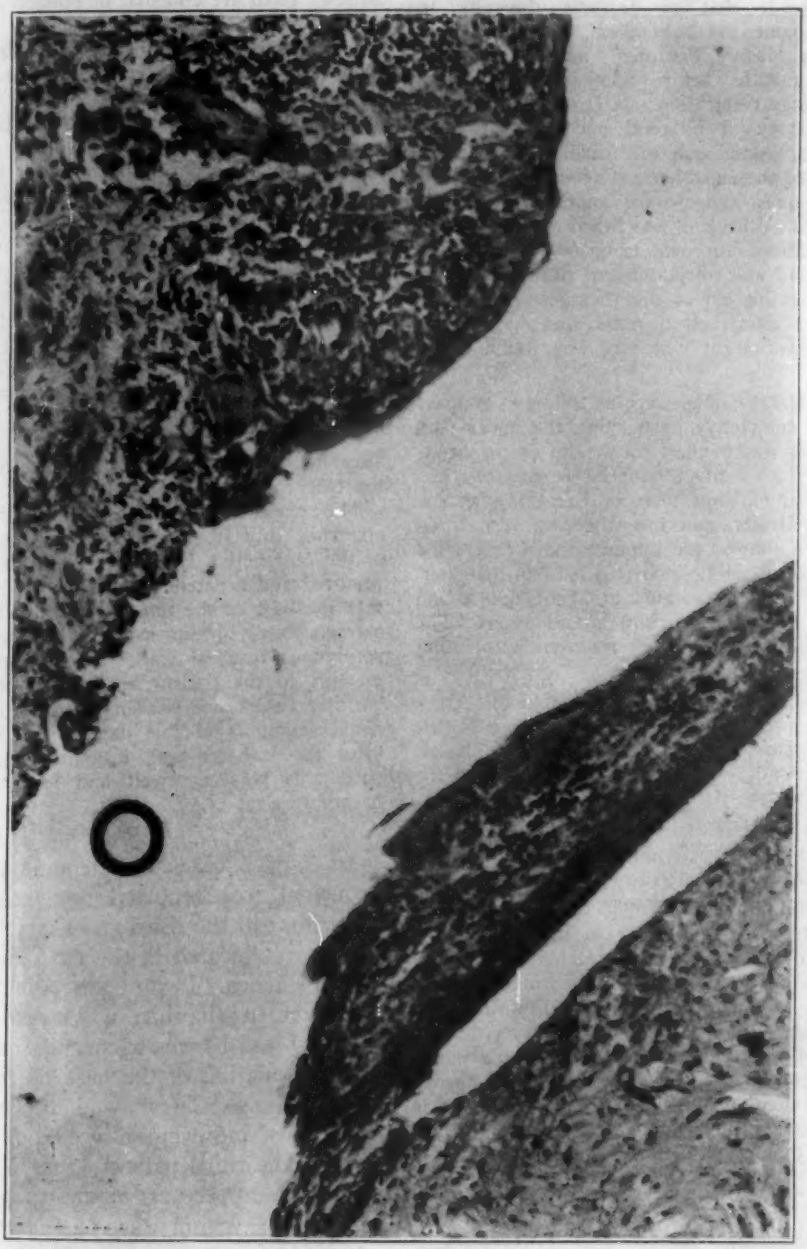

Fig. 11 (case 3).—Photomicrograph showing surface epithelium (×175). United States Army Medical useum negative no. 72509.

olied to the broad base of the growth in an attempt destroy it. The regrowth was so rapid that within week after removal the tumor had attained its preous size. Radon seeds were inserted into the tumor July 3, and high voltage roentgen rays were used, t both measures were completely ineffectual. Increasg bronchostenosis of the orifice of the upper lobe was dent.

The patient was eventually transferred to a hospital patients with chronic incurable diseases, where he

Study of the serial roentgenograms shown in figure 7 shows the changing picture observed from time to time. Obstruction was relieved by removal of the tumor only to recur rapidly with the regrowth of the tumor.

The following case is apparently unique. I have been unable to find any report of a case of a similar nature in the medical literature. There are occasional reports of adenoma arising from near

the carina or at the level of the tracheobronchial juncture but not in the upper part of the trachea as in this case.

CASE 3.—*(Adenoma of the Trachea).*—Mrs. C. H., a widow 61 years of age, born in Austria, was admitted to Beth David Hospital, New York, on Sept. 24, 1941. Her complaint was that of extreme difficulty in breathing, accompanied by headaches. She stated that she had been a "heavy breather" for a long time but that three months before admission she began to have wheezing respiration. Of possible significance was a history of bilateral pneumonia twenty-four years before. Arteriosclerotic cardiac disease and chronic phlebitis of the right leg were found on physical examination. Dyspnea and cyanosis of the lips and the tongue were noted. Wheezing was heard over the entire chest on auscultation. A roentgenogram of the chest showed angulation and displacement of the trachea (fig. 8). In reexamination of the roentgenogram after bronchoscopy, an intratracheal mass was seen to be present at the location in which it was found endoscopically.

The bronchoscopic findings were as follows: A mass was seen almost completely obstructing the upper part of the trachea. It was avulsed by the lip of the bronchoscope coming into contact with it. It was coughed out through the 9 mm. bronchoscope. Breathing became unimpaired immediately, and the wheezing respiration ceased. Inspection showed the tumor to have originated from a broad base about ¼ inch (0.6 cm.) in diameter, the center of which was located at about the second tracheal ring on the anterior right lateral aspect. The tissue from the base was partially removed with biting forceps.

The avulsed larger mass was submitted to the Army Medical Museum and the tissue from the base to the pathological laboratory of Beth David Hospital. Dr. M. Halperin, the pathologist of the hospital, submitted the following report on the tissue furnished him:

"The specimen consists of a fragment of tissue measuring 3 mm. in diameter, which represents a small part of the original specimen; the bulk of the specimen was not submitted to the laboratory.

"The specimen consists of closely placed solid alveolar structures, which are separated from each other by very thin fibrous tissue strands. Within the tumor alveoli the nuclei of the cells are pale and the cell outlines are well defined. Superficially the tumor is covered with small amounts of blood clots. In some areas the tumor alveoli are scattered in the more abundant fibrous tissue stroma. Occasional alveoli appear more cellular and more hyperchromatic, and the cells show slight spindling. The tumor cells in the more fibrous areas are distorted. Mitoses are not seen. . . ."

Dr. Halperin diagnosed the tumor as adenomatous neoplasm of the trachea. He stated that adenocarcinoma could not be ruled out since the main portion of the specimen, including the stalk, was not available for examination.

Col. J. E. Ash, curator of the Army Medical Museum, reported on the specimen submitted there as follows (figs. 9, 10 and 11):

"There are four sections. Each shows a bi... structure. The bulk of the lobular masses is c... posed of dense connective tissue, masses of h... and spaces resembling cholesterol slits. Between ... lobes there is a mass of tumor tissue that is po... demarcated and not at all encapsulated, composed ... fairly regular, moderately deeply staining epithelial c... Most of them are cuboidal or columnar, and freque... they have an alveolar, less frequently a microfollic... and glandular arrangement. No mitotic figures ... identified. Nucleoli are not prominent. If the ... arrangement of the tumor is correctly interpreted f... the sections, the surface of the tumor is ulcerated, b... covered on but a small portion by metaplastic epithel... showing some pseudoepitheliomatous hyperplasia. ... tumor proper is present at the base, thereby repres... incomplete removal. If our sections represent true c... sections of the tumor, a recurrence may be expe... It is assumed that the described hyaline masses re... sent degenerated portions of the tumor."

The diagnosis was adenoma of the trachea. Co... ants to whom the sections were sent by the m... reported as follows: J. W. Budd: "Epithelioma of ... tracheal mucosa, salivary gland tumor pattern." F. ... Harper: "Adenoma of the trachea." R. M. Mulli... "Adenoma of the trachea." T. B. Mallory: "Bronc... adenoma of the trachea." F. H. Foucar: "Adenom... the trachea." N. C. Foot: "Tracheal adenoma."

The patient underwent bronchoscopic examin... again on July 10, 1942 for a recurrence with a re... of the obstructive symptoms. The growth was gr... removed and a patent airway reestablished. It was ... that the base of the tumor could be eradicated thro... a tracheotomy opening with removal of a portion of ... contiguous tracheal wall. This procedure was rec... mended to the patient but so far has been decli... Further recurrence is anticipated, although a teleph... conversation as of this date, which is fifteen mo... after the last removal, brought the answer that ... patient is perfectly well and has no obstruction ... breathing.

SUMMARY

In 2 of 3 cases of adenoma of the trache... bronchial tree reported the tumor was in t... bronchus; in the third case, which is unique, t... tumor was located in the extreme upper porti... of the trachea. Two of the patients were midd... aged women; the third was an elderly man. O... patient (case 1) was apparently cured by remo... and coagulation of the base of the tumor. O... patient (case 2) showed repeated and ra... recurrence and eventually died of a cerebral ac... dent. The third patient (case 3), whose tu... was in the upper part of the trachea, has had ... recurrence within the past two years. T... patient refuses an external surgical attempt ... removal and is quite comfortable at the pres... time, one year and three months after the l... removal.

136 East Fifty-Seventh Street.

INTRACRANIAL METASTASIS OF CARCINOMA OF THE LUNG

WILLIAM S. TINNEY, M.D.,* AND HERMAN J. MOERSCH, M.D.
ROCHESTER, MINN.

The high incidence of intracranial metastasis cases of carcinoma of the lung has been long ognized. When such metastasis occurs, it only is of considerable prognostic importance may present an extremely difficult diagnostic blem that is deserving of emphasis. The t clinical manifestations of carcinoma of the g may be due to intracranial metastasis, which y give rise to a symptom complex closely ulating that produced by a rapidly growing highly invasive primary tumor of the brain. e differential diagnosis between a primary a secondary tumor of the brain is sometimes remely difficult and occasionally impossible.

The importance of the problem has been phasized by various observers. Ochsner and Bakey [1] in a review of 3,047 collected cases of rcinoma of the lung found that metastasis to brain had occurred in 16.5 per cent. Kolet-y,[2] Graham [3] and Arkin and Wagner [4] reported t intracranial metastasis occurred in 22 to 24 r cent of their cases of bronchiogenic carcinoma ile Olson [5] reported the extremely high inci-nce of 36.3 per cent.

King and Ford,[6] who reviewed 100 cases of rcinoma of the lung in which complete post-rtem examination of the nervous system was rried out, formed the opinion that carcinoma the lung metastasizes to the brain more fre-ently than any other type of carcinoma be-use the malignant cells can be carried directly the blood stream to the brain without passing through the capillary filter in the lungs. In several cases they noted, as had been previously reported, that evidence of intracranial metastasis appeared before any pulmonary symptoms developed. Grant,[7] Fried and Buckley [8] and King and Ford [6] have reported instances in which exploratory craniotomy was performed on patients who had primary carcinoma of the lung. In these patients, the pulmonary signs and symptoms were so meager that the pulmonary lesion was completely overlooked, while the neurologic signs were so prominent that a primary tumor of the brain was suspected.

Globus and Selinsky [9] expressed the opinion that metastatic tumor of the brain from carcinoma of the lung could be distinguished from primary tumor of the brain by the acute onset of disseminated and poorly localized signs of increased intracranial pressure without papilledema. Grant [7] likewise emphasized the rapid onset and progression of symptoms and particularly the occurrence of psychosis.

Because of the difficulty of differentiating between primary tumor of the brain and carcinoma of the lung metastasizing to the brain, and because of the frequency with which such metastasis occurs, King and Ford [6] and Craig, Woltman and Kernohan [10] advised that roentgenograms of the thorax be made as a routine in every case in which an intracranial or an intraspinal tumor is suspected and in all cases of unexplained stupor.

In order to determine whether any additional information might be uncovered in the early diagnosis of intracranial metastasis of primary carcinoma of the lung, we have reviewed 448 cases of carcinoma of the lung in which the diagnosis was confirmed by microscopic examina-

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2. Koletsky, S.: Primary Carcinoma of the Lung: Clinical and Pathologic Study of One Hundred Cases, rch. Int. Med. **62**:636-651 (Oct.) 1938.

3. Graham, E. A.: Primary Carcinoma of the Lung Bronchus, Ann. Surg. **103**:1-12 (Jan.) 1936.

4. Arkin, A., and Wagner, D. H.: Primary Car-noma of the Lung, J. A. M. A. **106**:587-591 (Feb. 22) 36.

5. Olson, K. B.: Primary Carcinoma of the Lung, n. J. Path. **11**:449-468 (May) 1935.

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7. Grant, F. C.: Concerning Intracranial Malignant Metastases, Ann. Surg. **84**:635-646 (Nov.) 1926.

8. Fried, B. M., and Buckley, R. C.: Primary Carcinoma of the Lungs: IV. Intracranial Metastasis, Arch. Path. **9**:483-527 (Feb.) 1930.

9. Globus, J. H., and Selinsky, H.: Metastatic Tumors of the Brain: A Clinical Study of Twelve Cases with Necropsy, Arch. Neurol. & Psychiat. **17**: 481-513 (April) 1927.

10. Craig, W. McK.; Woltman, H. W., and Kernohan, J. W.: Metastasis to the Central Nervous System from Carcinoma of the Lung, Am. J. Cancer **36**:12-24 (May) 1939.

tion of tissue obtained directly from the primary tumor. All of these cases were observed at the Mayo Clinic. Fifty-two, or 12 per cent, of the patients had symptoms referable to the nervous system. In 19 patients, or 4 per cent, the first symptoms were referable to the nervous system, and the patients had come to the clinic because of their neurologic symptoms. In 8 of these patients the clinical findings were such that a localized intracranial lesion was suspected, and 2 patients underwent exploratory craniotomy.

The group of 19 cases in which the first symptoms pointed to involvement of the nervous system was of particular interest because the differential diagnosis was extremely difficult. In 5 cases there were pulmonary symptoms only in the terminal stages of the disease. In 9 instances the first evidence of a pulmonary lesion was revealed on routine roentgenographic examination of the thorax. The diagnosis was confirmed by necropsy in 12 cases and by bronchoscopy in 7. There were several clinical features that were particularly characteristic in these 19 cases in which the initial symptoms were referable to the central nervous system. There was a rather high incidence of personality changes, such as loss of memory, disorientation and depression. These symptoms occurred in 8 cases. The most prominent clinical manifestation in 13 instances was the rapid progression of symptoms, more rapid than is generally found in cases of primary tumor of the brain. The average duration of life from onset of symptoms in the 448 cases of bronchiogenic carcinoma was fourteen and a half months, whereas in the group of 19 cases the duration was only six months. The type of carcinoma was of no significance in the incidence of intracranial metastasis.

Necropsy was performed in 70 of the total series of 448 cases. In 19 per cent of these 70 cases cerebral metastasis was found.

COMMENT

Because of the lack of specificity of pulmonary symptoms and the occasional absence of any symptoms referable to the thorax, the diagnosis of carcinoma of the bronchus not infrequently is extremely difficult. In some instances the problem is further complicated by early metastasis to the central nervous system, as exemplified by involvement of the nervous system in 52, or 12 per cent, of the 448 cases of pri carcinoma of the lung. The most interes cases were the 19, or 4 per cent, in which initial symptoms were referable to the ner system. In 9 of these cases a pulmonary le was not suspected until a routine roentgenog of the thorax was obtained. For this reason wish to stress the importance of careful phy examination, including routine roentgenogra examination of the thorax, in every case in wh an intracranial or intraspinal lesion is suspec In spite of this precaution 2 patients were s jected to exploratory craniotomy. It is o difficult and at times impossible to distinguis metastatic lesion from a primary tumor of brain or the spinal cord. The clinical manife tions in most of the patients suggested ch acteristically wide dissemination and rapid p gression of the lesions and included a rather h incidence of personality changes. Howe these clinical findings alone were not sufficie characteristic in any case to be of much assista in the differential diagnosis. We also wish emphasize that in our series of cases the type carcinoma of the lung was not a factor in incidence of metastasis to the nervous system

SUMMARY

There is a high incidence of intracranial met tasis in cases of bronchiogenic carcinoma. T first clinical manifestation of carcinoma of bronchus may be a symptom complex that clos simulates that produced by a rapidly grow and highly invasive primary tumor of the bra A series of 448 cases of microscopically pro carcinoma of the bronchus were reviewed, a in 52, or 12 per cent, of the cases there w symptoms referable to the nervous syste There were 19 cases, or 4 per cent, in which t symptoms referable to the nervous system w the presenting ones. A localized intracran lesion was suspected in 8 cases, and 2 patien underwent exploratory craniotomy. Necrop was performed in 70 cases, and in 19 per ce of these cerebral metastasis was found. thorough general examination including routine roentgenogram of the thorax should made in every case in which an intracranial an intraspinal lesion is suspected. The type carcinoma was not a factor in the incidence metastasis to the nervous system.

HEARING AIDS

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the close of World War I an estimated 00 aural casualties of all degrees of hearing irment returned to civilian life after dis- ge or resignation from the Armed Forces he United States. At the present moment e are six times as many men in the American ed Forces as there were in the last war and are scattered over the entire globe. On a ly numerical basis one may expect a quarter on aural casualties in this war. But there many other factors to be considered. The rds to hearing have increased to an im- surable extent. It can properly be assumed no combat aviator will return to civil life he war's end with normal hearing, despite fact that his induction into this branch of ervice required perfect hearing. Aural infec- and complications of contagion are un- tedly under better control, but acoustic ma, the effects of high altitude flying and its llary, acute pressure changes related to dive bing and fighting, and finally the remote ts of the administration of serum and anti- n and prophylactic drug therapy will increase ratio of aural casualties to a truly appalling nt. In the last war aural casualties were r cent of the total. This same numerical would give the quarter million estimated his war. That the actual number will be h greater can hardly be questioned.

he Army and Navy Surgeon General's Of- realize the importance of the problem and e taken steps to deal with the situation quately. The United States Veterans' Bureau reparing for this influx of soldiers who will returned to civilian life with a handicapping of hearing. Quite properly the various anizations whose primary purpose is caring hard of hearing and deafened civilians have n steps to deal with this serious postwar blem. Leagues for the hard of hearing established rehabilitation bureaus for handi- ped people will play an essential role in its l solution.

However, the entire situation is primarily a medical one and more specifically an otologic one. For this reason the American Academy of Ophthalmology and Otolaryngology has appointed a committee whose purpose shall be to insure every possible aid for these aural war casualties. This great group of men will be returned to their home communities with a serious handicap, namely, a hearing loss. They will have been cared for in an expert manner before discharge from the Armed Forces. But their care cannot stop at that point. On their return to civilian life an entirely new problem presents itself and an entirely new adjustment must be made.

It is rather an unhappy commentary on otologists and their concern for the deaf that a war must be the means of stirring them into decisive action. But unquestionably much good will result from this final effort provided they come to realize their responsibility to the deaf and hard of hearing.

Any disability whether incurred in civilian life or as a result of military service requires effort toward rehabilitation. The American Academy of Ophthalmology and Otolaryngology has among its appointed committees one which has to do with the prevention and amelioration of deafness and among the subcommittees one concerned with the rehabilitation of the deaf. According to Webster, one can be rehabilitated by "being restored to a former capacity," in this case hearing; or, as a second definition, rehabilitation means "to fit to make one's livelihood again." This discussion will be concerned primarily with rehabilitation.

The medical treatment of deafness is, as all otologists know, relatively unimportant and ineffective. Surgical treatment, if applied according to accepted precepts is from a numerical point of view insignificant. It is unlikely that any acquired war deafness will be amenable to surgery of any type other than that applied for the correction of chronic infection.

Civilians require rehabilitation now so that they may play their part in the general war effort. The aural casualties of this war are already being returned home as incapable of further military duty. The immediate handling of both types of hearing loss is identical. There

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is not time for medical treatment of uncertain value or promise. For the civilian, elective surgical intervention under war conditions is an improper hazard to undergo.

There is but one ready solution to this present and impending problem and that is the proper fitting of an adequate hearing aid. The Council of Physical Therapy of the American Medical Association through its Committee of Consultants approves or withholds approval of commercial hearing aids. The otologist is ethically bound to endorse only such approved aids.

Before proceeding further it may be well to modify the foregoing statements. The present situation is an emergency one which may last a matter of months or years. Every scientifically minded otologist, every research otologist, has before him the hope that new and immediately effective therapeutic technics may be developed. Were this not so his specialty would be a completely stagnant one. But for the immediate control of the present emergency it is felt that this exposition of therapeutic control of deafness is the only feasible one.

War casualties and civilian deaf persons require immediate help now. Otologists have more or less disregarded the particular professional obligation of intelligently advising their patients in the selection of an appropriate and efficient hearing aid. The reasons for this indifference are obvious. The fitting of hearing aids is time consuming, infinite patience is requisite, a high feeling of responsibility for psychologic reaction must be developed and finally the mundane factor of expense of equipment and meager financial return in relation to expended time and effort must be considered.

The Army and Navy and Veterans' Bureaus are primarily concerned with the profoundly deaf, that group whose welfare is not amenable to aids of hearing, who depend on lip reading and speech training for their social and economic rehabilitation. The otologist's chief concern is the soldier who is discharged or who resigns from the Armed Forces with a handicapping loss of hearing. It is safe to assume that all surviving combat aviators will be in this group—innumerable men who have been exposed to the incessant acoustic trauma of gun fire of whatever type. Regardless of the cause, the forgotten man will be the one with a loss ranging from 35 to 60 decibels in the speech frequency range. At the present time the persons in the upper level of this group, seriously handicapped as they are, do not fit into the rehabilitation scheme. They present an important challenge to all otologists. The latter must be prepared to advise intelligently as to the pros and cons of hearing aids.

TYPES OF HEARING LOSS

Fundamentally there are three types of h ing loss for every level of impairment. Bea public health survey[1] simply confirmed grand scale what had been common knowl to all otologists for many years. He repo subjective disability as related to degree hearing loss. He[2] further made recomme tions for hearing aids as related to these spe losses. Theoretically these recommendat are sound. Practically they are of no valu cause at no point is the psychology of deaf considered. The psychologic factors are essence of all compensation by hearing aids.

In the first type of hearing loss the imp ment is uniform in degree throughout the au metric range; in the second it is accentuate the higher frequencies (nerve deafness); in third the hearing for low frequencies is n seriously impaired, as in chronic suppuratio the middle ear. These factors are all mo fied by the duration of impairment, the de of impairment and the type and by the age, sex and the social conditions of the patient. hard of hearing or deaf person will always an individual—he can never be treated simply a member of a group.

HEARING AIDS

For practical reasons the present discuss will be concerned only with vacuum tube hear aids of the air and the bone conduction ty Approximately twenty-five such aids have be accepted by the Council of Physical Thera For the most part unaccepted aids would n be generally used for the very reasons that th were not approved. However, the otolog should be familiar with the accepted list a should see to it that his patient is safeguarde

Modern vacuum tube hearing aids are fai uniform in construction and design, the princi differences being in size, weight and appearan also in size and weight of batteries.

The essential parts, all miniature, consist o microphone, an amplifier or transmitter with t or three midget vacuum tubes, condensers, r sistances, etc., a receiver which attaches to fitted ear piece and a battery pack consisti of an A battery of 1½ volts and a B batte of voltage ranging from 20 to 45 volts, depen ing on the type and the power of the instrume

1. Beasley, W. C.: The General Problem of De ness in the Population, Laryngoscope **50**:856 (Sep 1940.

2. Beasley, W. C.: Partial Deafness and Hearin Aid Design: I. Characteristics of Hearing Los Various Types of Deafness, J. Soc. Motion Pict Engin. **35**:59 (July) 1940.

he amplification supplied by the aid affects audiometric frequencies involved principally e speech range, namely 256 to 4096 cycles. modifications in design and construction a in degree of selective emphasis can be de- ed. This is usually in the higher frequency e. The amplification of low frequency tones odified at all, is usually in the direction of pression. Aids are available with a fixed fre- cy characteristic or are equipped with a tone rol which modifies to a certain extent the acter of the instrument's response.

FITTING OF HEARING AIDS

he fitting of hearing aids must be a function he otologist. It is essential, therefore, that members of this medical specialty acquaint nselves with the intricacies, and the word sed advisedly, of this relatively new adjunct heir armamentarium. This is no easy task, it is an essential one and at the present t be adequately met. Space here does not mit of discussion of any phase of the problem r than the actual methods of fitting hearing . The deaf or hard of hearing subject is cerned only with an instrument which will ease his ability to understand speech. The jority can hear speech as delivered, but it is aningless to them. Therefore an adequate ring aid must supply sufficient compensation a given deficiency to restore the vital quality ntelligibility. All other factors are essentially levant.

Methods of Determining Speech Reception vels.—In this discussion the establishment of ring levels based on uncontrolled spoken whispered voice tests will be immediately missed as inadequate, unreliable and in the e of compensation utterly unfair. Such a test be of only the slightest scientific conse- nce when the spoken voice is presented to the ener in the same degree of controlled in- sity as are individual frequencies by the prop- calibrated audiometer. Here again, as in king discrete frequency audiograms, a sound- of room is the ideal situation. If it is unavail- e, a sound-treated room should be provided. Three methods of presentation of spoken ce are available: First, spoken voice con- lled electrically with an accurate attenuating tem and presented indirectly to the listener means of a high fidelity loud speaker.[3] Sec- d, spoken voice presented through an elec- ally controlled circuit but projected from nograph records as the sound source. Third, ken voice presented through a roughly cali- brated nonelectrical attenuating system the general intensity levels of which can be reasonably well estimated.

Method 1. This method requires equipment which will be found in only a few medical or acoustic laboratories. There must be a soundproof room where speech can be presented to the subject by means of a loud speaker. The intensity of voice must have a calibrated attenuating control capable of modifying loudness in steps of 5 decibels or less over a range of 50 decibels above normal threshold. Beyond this point most electrical equipment tends to overload and distortion is introduced.

A list of sentences is read by a speaker in a soundproof booth through a microphone and an amplifying system. The intensity of the speaker's voice is modulated by observing the excursion of a decibel meter indicator needle. As in standard audiometry the subject records accurate understanding of speech by establishing a level of accuracy of at least 60 per cent. Here accuracy is determined by repeating the presented sentences verbatim instead of pressing the signal for heard or not heard as in the audiometric examination.

Sufficient time is allowed after the presentation of each sentence for the subject to repeat what he has heard without undue hurry. Each sentence has a key word or words which are critical in the correct repetition of the sentence. Interestingly enough this method is accurate to plus or minus 1 decibel and does not introduce any element of deterioration in production and it is completely flexible, both of which factors must be taken into account in method 2.

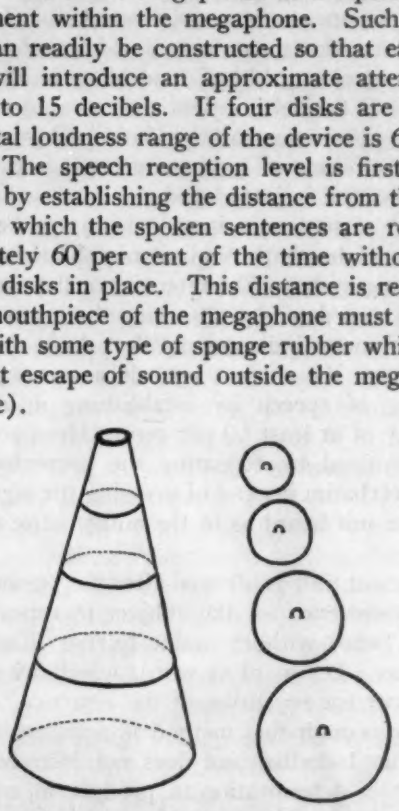
Method 2. In this method phonograph records are used as the source of speech, but the general electrical system and the physical surroundings are essentially the same. This method requires one less in the examining personnel but is entirely inflexible as to timing and immediate choice of sentences presented and is highly susceptible to deterioration of the records due to repeated playing. The sentence lists used are those devised by the Bell Telephone Laboratories and comprise 50 lists of 50 sentences, each of which would involve a considerable number of records.

Method 3. If none of the aforementioned equipment is available, a reasonably accurate test can be carried out in the following manner. This test is based on the assumption that the normal spoken voice of any person is more accurately controllable than an attenuated voice such as the whispered voice.

Any standard 2 foot (60 cm.) megaphone increases the intensity of voice spoken into it by approximately 20 decibels. If normal speech is of the order of loudness of 40 decibels above

3. Hughson, W., and Thompson, E.: Correlation Hearing Acuity for Speech with Discrete Frequency iograms, Arch. Otolaryng. **36**:526 (Oct.) 1942.

threshold and to this is added an additional 20 decibels, few impaired hearing levels susceptible to artificial aid would fail to be reached through such a system. Attenuation in the system can be introduced by a series of felt disks graded in diameter, the diameter of each disk corresponding to that of the megaphone at the point of its placement within the megaphone. Such a system can readily be constructed so that each felt disk will introduce an approximate attenuation of 10 to 15 decibels. If four disks are placed, the total loudness range of the device is 60 decibels. The speech reception level is first determined by establishing the distance from the subject at which the spoken sentences are repeated accurately 60 per cent of the time without any of the disks in place. This distance is recorded. The mouthpiece of the megaphone must be circled with some type of sponge rubber which will prevent escape of sound outside the megaphone (figure).



The standard commercial megaphone 2 feet (60 cm.) long. Disks are spaced 6 inches (15 cm.) apart and introduce attenuation of approximately 12 decibels for each disk, giving a total attenuation for spoken voice of 40 decibels. The felt is standard ½ inch (1.27 cm.) material stiffened by means of wire screening.

Fitting the Aid.—In any one of the three systems described in the foregoing section an unaided speech reception level is determined. Air and bone conduction audiograms have been made, and otologic examination has determined whether or not the impairment is complicated by chronic suppurative otitis media or not. In general this is the prime deciding factor between the use of an air or a bone conduction hearing aid.

Hearing aids are constructed either with a specific frequency characteristic, i. e., with a flat, a high frequency or a low frequency emphasis, or with tone controls by which the characteristic of the instrument may be changed to fit the specific need. In addition several aids supply receivers which are supposed to contribute to intensity as well as frequency response. If the receiver is a crystal one, the larger it is the greater will be the high frequency emphasis, particularly in the neighborhood of 3000 cycles. T is an important factor.

It is quite natural that an instrument c plementing the audiometric hearing loss w be the one first chosen for test. However, may not be the one finally selected. The kn over-all gain for speech must be a matter record.

Whichever of the three testing methods li in the foregoing section is used, the exten gain over unaided hearing will be the mea of each individual instrument's effectiveness the electrical methods the addition of attenu is the measure of acoustic gain; by the m phone method the number of felt disks w can be introduced into the device and still h intelligibility of speech will give a rough e mate of the aid's amplification.

Many factors are involved in this problem the war casualties old age — the most diffi group of all to fit — will not be a factor. T ex-serviceman will not be particularly concer with the appearance or the size of the instrum and batteries so long as the desired results obtained. These factors are often critical in fitting of aids for civilians and particularly, course, women.

But in the two groups there is one sine q non. No hearing aid of either the air or the bo conduction type should be considered unless gives a gain of 30 decibels in speech recept regardless of the established unaided hear level.

As an addendum it can be stated that vacu tube aids are vastly superior to carbon mic phone instruments. Air conduction aids basically far superior to bone conduction a which represent a hearing efficiency under o timum conditions of 30 decibels below that normal air conduction and which have a fr quency range limit of less than 3000 cycles, wh is the critical level for normal reception of spee

Never prescribe a bone conduction aid if it possible to use an air conduction instrume

Ear Molds.—For air conduction a well fit ear piece is essential. Even with the best inst ments the "feedback squeal" will develop if t receiver and earmold fit is not an adequat close one. Many hearing aid agencies a equipped to make plaster casts of the exter ear and cartilaginous canal. Dentists also p form this service. The latter are undoubted better equipped provided the otologist has m an otoscopic examination and can give a sat factory report on the patency of the exter canal and the intactness of the tympanic me brane. Any ear mold made without such supe vision is a direct, specific and unauthoriz

mption of a medical prerogative and should usceptible to action under the established l rules of medical practice.

SUMMARY AND CONCLUSION

he present world situation poses two major blems for the otologist: first, the immediate f of civilian hearing impairments as an aid he war effort and, second, the care and re-bilitation of aural casualties of the war who returned to civilian life. It is true that many hese veterans will still be under the direction control of the Veterans' Bureau, but there be a highly significant number who for e reason will be without medical advice control. American otologists must assume responsibility in the broadest possible man- They must familiarize themselves with the truction and performance of hearing aids pted by the Council of Physical Therapy of the American Medical Association. They must conform in their technic of fitting aids to some more or less uniform standard of procedure. Their records of successful performance will be a matter of great importance, as will be their records of failure.

The medical prerogative in this relatively new field should be jealously guarded. Hearing aids are manufactured and sold primarily for profit. Variations in sales technic and accepted advertising methods are designed to increase this profit through greater volume of sales. Beyond the fact that their product should be an excellent one the hearing aid companies should have no particular obligation or concern. Otologists have been laggard in assuming responsibility in this important part of the therapy of deafness. That they must assume it now permits of no discussion.

VASCULAR POLYP OF THE VOCAL CORD

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Vascular polyp is the most common of growths occurring on the vocal cords. It is known variously as hemangioma (Kramer and Yankauer [1]), varix (Faunce [2]; Imperatori [3]), inflammatory tumor (Tucker [4]), cavernous papilloma (Hooper [5]), angioma (Chiari [6]), hemangiectatic fibroma and papilloma (Eppinger [7]), capillary angioma (Schwartz [8]) and pseudoangioma (New [9]). I prefer the name vascular polyp. Vascular polyp is not a true growth but is inflammatory in nature. It is the result of trauma in the form of strain or abuse in the presence of chronic inflammation of the mucous membrane and the immediate subjacent tissue. Because vascular polyp is so distinctive, it is difficult to confuse with other benign growths of the larynx. It frequently is called papilloma or fibroma; this is evidence of the perpetuation of erroneous nomenclature.

It is likely that vascular polyp has been occurring ever since man first found his voice. From a perusal of the literature of the prelaryngoscopic era one gets the impression that all conditions which might cause suffocation were included under the general heading of polyp of the larynx. In the writings of Ehrmann [10] (1844) and Green [11] (1852) there is mentioned a series of cases, all of which were considered as cases of laryngeal polyp. All the patients w threatened with asphyxia; 3 were saved by eration.

After the invention of the laryngeal mirror Garcia (1855) there was a better understand and therefore a finer differentiation of the n plasms and other lesions found in the lary Yet it was not until fairly recently that a c understanding and a fairly accurate diagno became the rule.

The removal of a tumor of a vocal cord thro the natural passages was recorded as early 1750 by Kaderik.[12] Fauvel [13] is credited w having been the first to recognize what w probably a vascular polyp by mirror examinati of the larynx. He found a polyp "in the fi ward part of the vocal cord of a man"; cavernous structure of the polyp was demo strated microscopically. For the removal laryngeal growths Desault [14] recommended lary gotomy in 1813. Twenty years later Brauers attempted the removal of a polyp by this mea Three years after that, in 1836, Regnoli [16] moved a neoplasm through the mouth after fi performing tracheotomy. In 1844 Ehrmann also removed a polyp in this manner. By 18 Mackenzie [17] was able to collect 28 cases in whi thyrotomy had been performed for a growth the larynx. In prelaryngoscopic days and some time afterward, laryngeal growths were r moved by means of thyrotomy. This operati was used until the establishment of indirect pr cedures for the same purpose by the pioneers laryngology. On rare occasions a laryng polyp has been encountered in the dissecti room. It was only after Turck and Czermak h popularized the use of the mirror that repor

1. Kramer, R., and Yankauer, S.: Hemangioma of the Larynx, Laryngoscope **34**:405 (June) 1924.

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11. Green, H.: On the Surgical Treatment of Polypi of the Larynx, and Oedema of the Glottis, New York, G. P. Putnam, 1852, p. 124.

12. Kaderik, cited by Gottstein, J.: Diseases of t Larynx, translated by W. M'Bride, Edinburgh, W. A. K. Johnston, 1883, p. 124.

13. Fauvel, C.: Polype du larynx, Bull. Soc. de ch de Paris **3**:404, 1863.

14. Desault, P. J.: The Surgical Works, or Stat ment of the Doctrine and Practice of P. J. Desa by Xavier Bichat, translated by E. D. Smith, Phi delphia, T. Dobson, 1814.

15. Brauers, cited by Mackenzie,[17] p. 3.

16. Regnoli, cited by Mackenzie,[17] p. 3.

17. Mackenzie, M.: Essay on Growths in the Laryn Philadelphia, Lindsay & Blakiston, 1871.

umors such as that under consideration be- to appear in the literature.

teady progress has been made in the accuracy he diagnosis and the management of laryngeal wths since the turn of the century. The ex- nt articles by Kramer and Yankauer,[1] Im- tori,[3] New and Ehrich,[9] Lallemant, Delarue Leroux-Robert [18] and Friedberg and Segall,[19] ng others, have contributed materially to this gress. Moore [20] reviewed the literature from 1 to 1922 and reported 68 cases. Kramer and nkauer [1] reported 14 cases which they con- red instances of hemangioma. Imperatori [3] cribed a series of 14 neoplasms, to which he e the name varix. Jackson [21] stated that he seen 227 in forty years, while New and ch reported on 722 noncancerous tumors of larynx, 332 of which were inflammatory. nce [2] made a histologic study of 16 such nors; he called these growths varices. More ently Robb [22] wrote an interesting article the same subject. This paper is based on cases of vascular polyp of the vocal cord ich were under the author's observation.

The work of Chiari,[6] Reinke,[23] Hajek [24] and ldapfel [25] has afforded a better understanding the anatomic changes which precede the ap- rance of vascular polyp. Some form of vocal se in the presence of inflammation results in production of this structure. Most frequently inflammation is chronic and can be traced to infection of the nasal structures. Normally, submucous space in the region of the free ge of the vocal cord is limited. As a result of lammation and the presence of edema the sub- cous space is increased. It is in the presence such increase that the attachment of the cous membrane is less secure. Under such nditions, when the vocal cord is traumatized misuse or abuse, an extrusion or evagination of a localized portion of mucous membrane and submucosa may occur at or near the edge. Veins which exist in the submucosa are carried along with the extruded or evaginated section of the cord. During this process some of them may be ruptured. This is the mechanism of the production of the vascular polyp.

Vocal abuse usually consists of sudden exaggeration of vocal sounds, such as screaming, screeching or yelling. It also includes improper use of the vocal cords in the projection of the voice. It is for this reason that such growths tend to develop in singers, lawyers, speakers, salesmen, actors and teachers. Persons who are nervous or emotional are also subject to the formation of vascular polyp. Three of the patients in this series were afflicted with a mild form of Parkinson's disease, which was responsible for improper production of voice.

INCIDENCE

The incidence of localized vascular polyp of the vocal cord cannot be accurately established. This is due to difference in the interpretations of the histologic structure and to confusion of the terms applied to it.

A majority of the patients are males, probably because males are more often employed in fields in which the voice is likely to be abused. Seventy per cent of the 110 patients in this series were males; 75 per cent of a large series of inflammatory laryngeal tumors reported by New and Erich occurred among males. The higher incidence of these growths among males might be in some degree due to the greater amplitude and intensity of the vibrations of the vocal cords.

In this series of cases the ages ranged between 17 and 70.

Analysis of One Hundred and Ten Cases According to Age

Age Group	Cases
11-20	1
21-30	23
31-40	35
41-50	35
51-60	10
61-70	5
71-80	1
Total	110

Vascular polyp is never seen in the vocal cords of children. The explanation probably lies in the fact that children rarely suffer chronic inflammation of the vocal cords or that the structure is different from that of the adult. Another reason is that there is greater tolerance for vocal abuse because of the greater resiliency of tissue. Those between the ages of 25 and 50 are most susceptible. Localized vascular polyp

8. Lallemant, M.; Delarue, J., and Leroux-Robert, Laryngeal Polypi, Ann. d'oto-laryng., January 1939, 9.

9. Friedberg, S., and Segall, W. H.: The Patho- ic Anatomy of Polyps of the Larynx, Ann. Otol., in. & Laryng. **50**:783 (Sept.) 1941.

0. Moore, I.: Further Remarks on Angiomata of Larynx, J. Laryng. & Otol. **38**:57 (Feb.) 1923.

1. Jackson, C., and Jackson, C. L.: Benign Tumors the Larynx, in Jackson, C., and Coates, G. M.: The se, Throat and Ear and Their Diseases, Philadelphia, B. Saunders Company, 1929.

2. Robb, J. M.: Varix of the Vocal Cord, Ann. ol., Rhin. & Laryng. **47**:522 (June) 1938.

3. Reinke, F.: Untersuchungen über das menschliche mmband, Fortschr. d. Med. **13**:469, 1895.

4. Hajek, M.: Beiträge zur Anatomie der Stimm- gen, Ztschr. f. Hals-, Nasen- u. Ohrenh. **13**:161, 1925.

5. Waldapfel, R.: Pathology of the Subepithelial einke's) Layer of the Vocal Cords, Ann. Otol., Rhin. Laryng. **49**:647 (Sept.) 1940.

is rarely seen after 60, although Sommerbrodt [26] reported the case of a clergyman who was 80 years of age.

GROSS APPEARANCE

Vascular polyp is usually single but may occur in two sites in the same larynx. Among the 110 cases were 8 in which a growth existed on each

ber of vessels or vascular spaces present and thickness of the mucosal covering. The loca of vascular polyp is confined almost exclusi to the anterior third of the vocal cord. I series of 110 cases, of which 8 were doc comprising a total of 118 growths, a polyp located in the extreme anterior commissure in

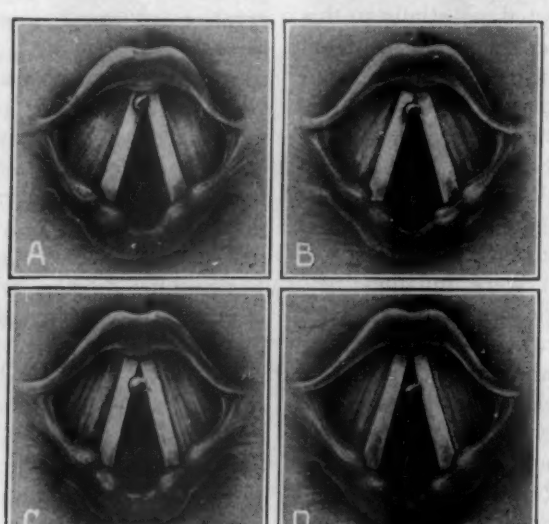
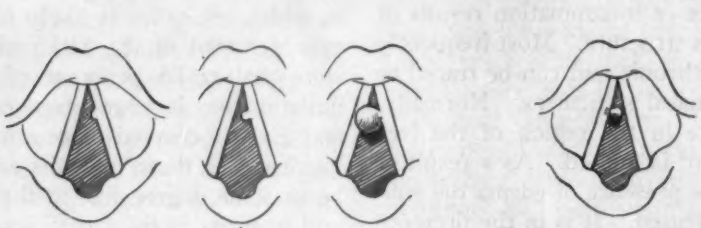


Fig. 1.—Most frequent locations of vascular polyps.



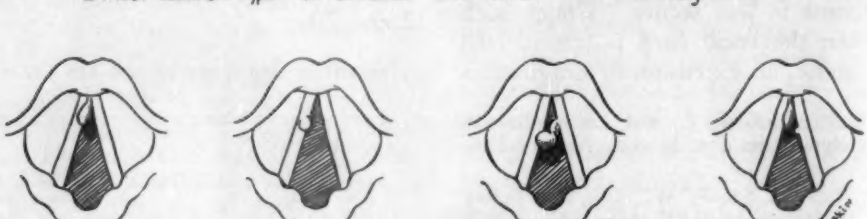


Fig. 2.—Various forms of vascular polyps.

cord. The size varies from that of a pinhead to that of a mass 2.5 cm. in length and 6 mm. in diameter. The shape may be spherical, ovoid, cylindric, piriform or nipplelike.

The color is reddish blue, grayish blue, cherry red, purplish or grayish, depending on the num-

In 68 it was located at the point of juncture tween the anterior and middle thirds of the vo cord, while in 31 it was found between these t locations. Only in 2 cases was a polyp fou located posterior to the anterior third of t vocal cord (fig. 3). In 69 cases a polyp w found on the right vocal cord and in 49 on t left. Both cords were involved in 8 cases.

26. Sommerbrodt, J.: Drei Operationen von Kehlkopfpolypen, Wien. med. Presse **13**:569, 1872.

the polyp is recent, it is quite soft. As time es, however, it becomes more firm and the ring more friable. The extent and the rapidf these changes depend on the amount of ation and trauma to which the growth is ected during phonation or other acts requirclosure of the glottis.

he attachment is usually sessile. When the p is pedunculated, increase in its size and ght, together with increased tension on its chment, causes a thinning of the pedicle. rare instances the pedicle may become so ned that the tumor becomes detached and is elled.

from the adjacent healthy mucous membrane. The covering of a polyp having these characteristics is readily ruptured by the grasp of forceps, in much the same way as the skin of a grape that is being squeezed. The epithelial covering is virtually a capsule. The basement membrane is well defined and always intact. Larger growths may suffer a localized loss of epithelial covering as a result of repeated contact with or friction against an apposing surface. In such cases one may encounter a streak of blood in the larynx or in the expectoration.

The submucous connective tissue may be loose or firm. The degree of firmness varies with the

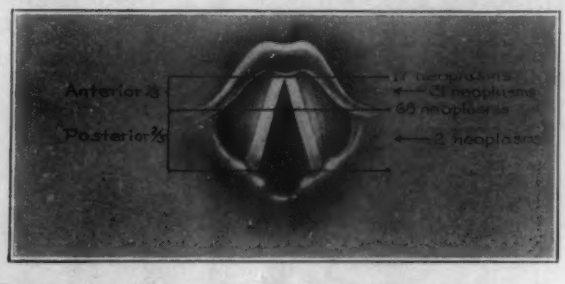


Fig. 3.—Location and distribution of 118 vascular polyps.

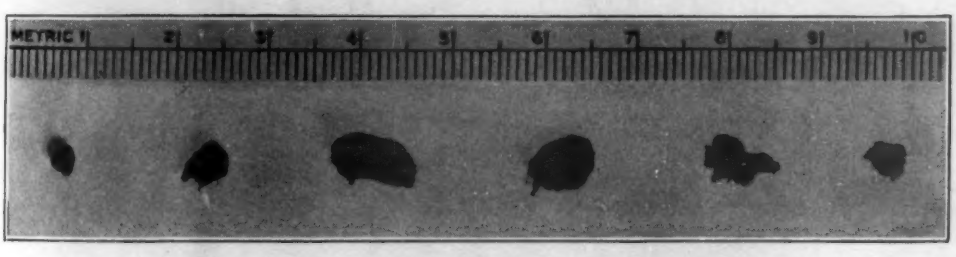


Fig. 4.—Photograph of vascular polyps after removal.

HISTOLOGIC APPEARANCE

he histologic structure of vascular polyp, algh characteristic, presents many variations. e articles of Lallement, Delarue and Lerouxbert [18] and Friedberg and Segall [19] on this ject are noteworthy. The mucous membrane be normal or slightly thickened at the time growth first appears. It becomes thicker or ner, depending on the duration of the growth its reaction to the mechanical irritation to ch it has been subjected. With the passage ime the epithelium becomes flatter; the orig- columnar cells become cuboidal and eventu- squamous. The mucous membrane is thicker the unexposed sides than on the exposed sure of the growth. The epithelium of a longnding vascular polyp becomes thinned, kerazed and sclerosed; for this reason it can be ached readily from the rest of the growth and age of the polyp and the amount of irritation to which it has been subjected, also with the amount of edema present. Edematous infiltration may be quite pronounced, the extent depending on the intensity of the inflammatory reaction. The submucous space, which is normally small, is tremendously increased by the edema. Reinke [23] Chiari,[6] Hajek,[24] Waldapfel [25] and others have emphasized the significance of this edematous space in relation to the genesis of vascular polyp. The greater part of a cross section of a vascular polyp may be occupied by a cystlike space filled with blood, around which there are hyaline changes. In other parts of the stroma, the tissue is homogeneous, having the appearance of myxoma, while elsewhere fibroblasts predominate. In some growths numerous spaces lined with endothelial cells and filled with decomposing blood are encountered. Well formed blood vessels are

seen in long-standing growths; in the more recent ones blood spaces are common. At times fibrin and an organized blood clot can be discerned; plasma and round cells abound, and an occasional leukocyte may be present. After a relatively long period of time the blood spaces fuse into a formless homogeneous mass in which fibrin, platelets, nuclei of leukocytes and red blood cells lose their identity.

Reports from several pathologists are presented to illustrate the various interpretations

thin superficial keratinized layer is present. demarcation between the basal layer and the stro sharp. The main internal portion of the mass co of edematous stroma and large spaces containing and small thrombi. The spaces are often devoid endothelial lining (i. e., extravasated blood). In places an endothelial lining is seen, and the sub stroma contains numerous pigment-laden cells. morphonuclear leukocytic infiltration is slight. some areas, hyaline fibrosis and numerous small emic blood vessels are seen. Diagnosis: F hemangioma of the vocal cord."

Doctor C: "Section shows a minute fragme issue with lining columnar and stratified colu

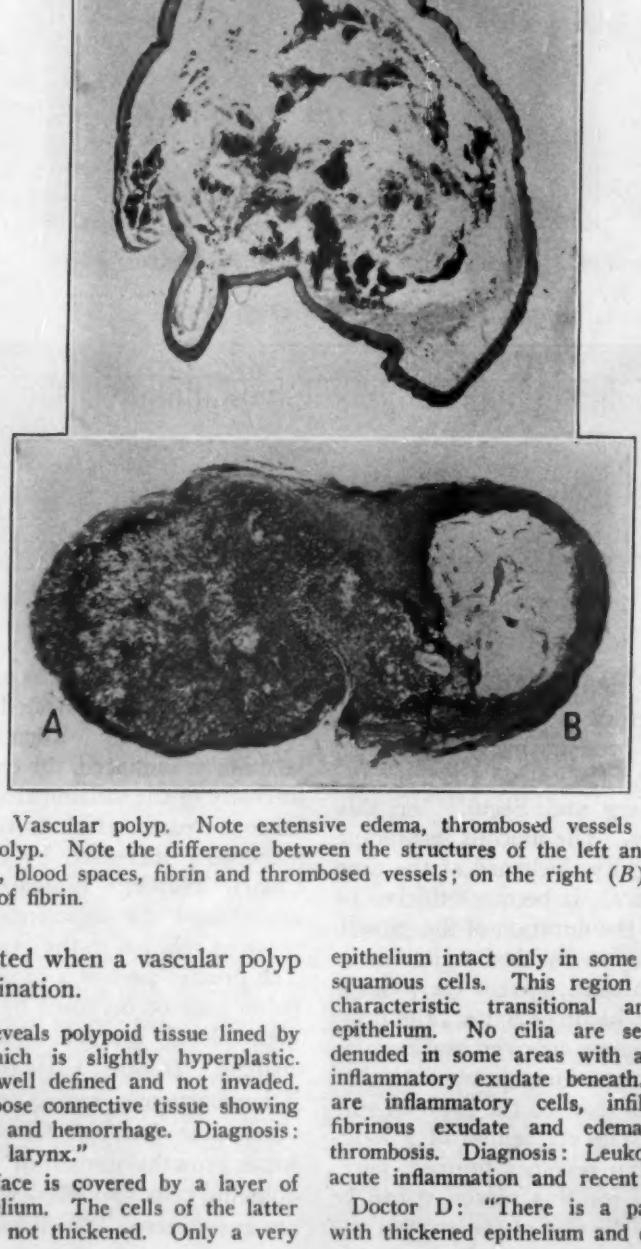


Fig. 5.—Upper part: Vascular polyp. Note extensive edema, thrombosed vessels and extravasated b Lower part: Vascular polyp. Note the difference between the structures of the left and the right half: o left (*A*), thinner mucosa, blood spaces, fibrin and thrombosed vessels; on the right (*B*), pachyderma, myxo tous stroma and strands of fibrin.

which are to be expected when a vascular polyp is submitted for examination.

Doctor A: "Section reveals polypoid tissue lined by squamous epithelium which is slightly hyperplastic. The basal membrane is well defined and not invaded. The stroma consists of loose connective tissue showing myxomatous degeneration and hemorrhage. Diagnosis: Myxomatous polyp of the larynx."

Doctor B.: "The surface is covered by a layer of stratified squamous epithelium. The cells of the latter are regular; the layer is not thickened. Only a very

epithelium intact only in some areas. One area s squamous cells. This region merges with a zone characteristic transitional and stratified colu epithelium. No cilia are seen. The epithelium denuded in some areas with a granulomatous type inflammatory exudate beneath. In the stroma t are inflammatory cells, infiltration, vascularizati fibrinous exudate and edema. Some vessels s thrombosis. Diagnosis: Leukoplakia with chronic acute inflammation and recent ulceration."

Doctor D: "There is a papillary growth co with thickened epithelium and composed of degener

us tissue and distended vessels. Diagnosis: ofibroma."

ctor E: "The nodule is covered with a layer of al squamous epithelium. It is composed of con- ve tissue in which there are a number of large s lined by endothelium. It has the structure of ge hemangioma. Diagnosis: Benign hemangioma."

is to be noted that vascular polyp is con- ntly covered with mucous membrane, that it esents outpouching of the original mucosa, some of the submucosal elements included, that these submucosal elements undergo cer- changes as a result of duration and local ma.

SYMPTOMS

oarseness, vocal fatigue and the sensation of reign body are the symptoms of vascular p of the vocal cord. Cough and dyspnea are mmon. The first and at times the only ptom is hoarseness. The degree of hoarse-

g. 6.—Vascular polyp. Note the area of hyalini- n at the upper center and the thrombosed vessels empty vessel traversing the lower center of the

depends on the extent of the interference the approximation of the vocal cords; the ter the interference, the greater the degree oarseness. A small growth influences the less than a large one; a growth situated riorly interferes more with vocal function. edunculated polyp attached to the edge of the l cord drops down and is frequently forced ard on the surface of the cord by the expira- effort incidental to phonation. When this e case there is no interference with the pro- ion of sound. In addition, the patient may onscious of the fact that the growth drops into the glottis after he uses his voice.

ocal fatigue is a frequent symptom. It is ed by the extra effort required for phonation. l cases in which there is poor approximation e vocal cords, normal vibration and tension are unfavorably affected, causing the abductor and tensor muscles to be overworked. The extra effort causes the voice to spend itself within a short time, and the muscles to become fatigued and sore.

The sensation of a foreign body and the irritation of a moving tumor mass may cause the patient to cough. This is more likely to occur

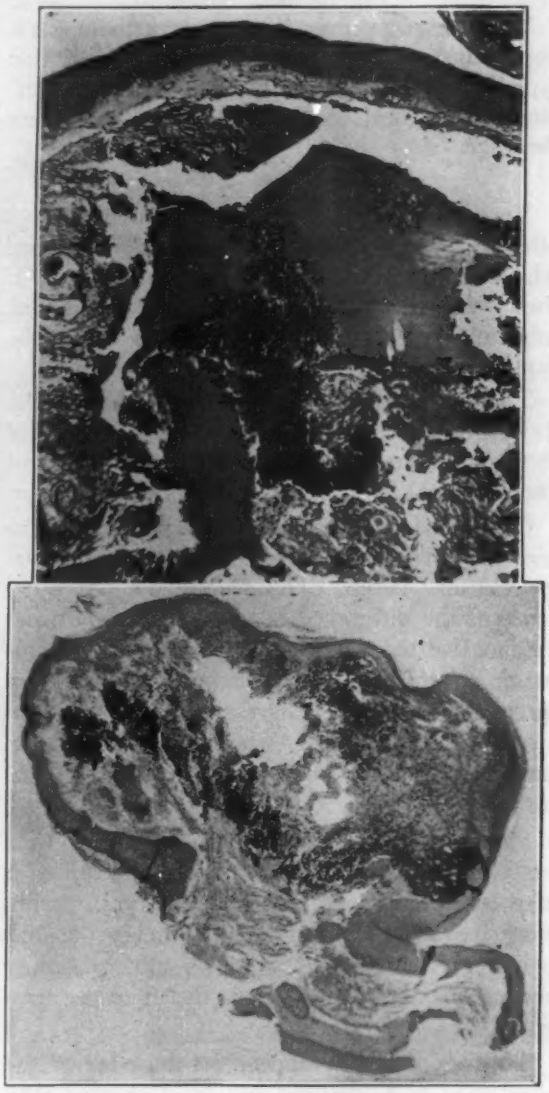


Fig. 7.—Upper part: Section of vascular polyp magnified to illustrate hyalinization of a vascular space. Note blood cells lying free in the center of the field. Lower part: Vascular polyp. Note thrombosed vessels, extravasated blood, fibrin and some edema.

when a pedunculated growth moves back and forth between the supraglottic and infraglottic regions during active respiration.

Dyspnea may result from a polyp of unusual size. Fortunately, the growth rarely reaches a size sufficiently great to endanger the airway.

It becomes larger in the presence of infection of the respiratory passages. The dyspnea may be both inspiratory and expiratory but is more often the former. In 1 case of this series tracheotomy was necessary. The patient was a woman 42 years of age who had a fairly large vascular polyp. She contracted an infection of the upper respiratory tract which caused her laryngeal tissues to swell to such an extent that the resulting dyspnea made tracheotomy necessary.

Vascular polyp may be present without causing symptoms. This is especially true of that type which is occasionally located on the surface of the vocal cord and does not hang over the edge.

DIFFERENTIATION

Vascular polyp must be differentiated from other small, localized growths of the vocal cord which have a mucous membrane covering. Of these, the vocal nodule, carcinoma, papilloma, tuberculoma and edematous fibroma should be kept in mind.

The vocal nodule is readily distinguished by its constant location at the point of juncture of the anterior and middle thirds of the vocal cord (nodal point), its small size and its whitish color.

Carcinoma in a very early stage situated on the anterior third of the vocal cord may be mistaken for vascular polyp. This should not occur because of the difference in texture and color and the fact that there is infrequently a mucous membrane covering. The infiltrative character of carcinoma gives the impression of a neoplasm merging with the cord, whereas vascular polyp stands out boldly and distinctly from the vocal cord.

Papilloma would be difficult to confuse with vascular polyp because its surface is irregular; it may be located anywhere in the larynx, occurs more frequently in childhood, is never purplish red, nor is it covered by a smooth, glistening mucous membrane. There is usually more than one.

Tuberculoma may appear on the edge of the vocal cord as a localized neoplasm covered with mucous membrane of a dull red color. It rarely assumes the distinctly round or ovoid form of vascular polyp. There will be cases, however, in which only microscopic study will reveal the true nature of the lesion. In most cases tuberculous disease which simulates a neoplasm of the vocal cord occurs in the form of granuloma, which is not covered by mucous membrane.

Edematous fibroma may occur anywhere along the edge or the surface of one or both vocal cords; it is most frequently bilateral. It is polypoid, flat and extensive, usually larger than a vas polyp and pearly gray.

One should bear in mind that vascular p has a distinctive color—cherry red or pur red. It does not increase in size except in presence of inflammation; it does not infil and does not extend beyond the original si attachment.

When examining a patient with vascular p one may observe that the opposite cord is peremic or hypertrophic; or a keratotic fibromatous nodule may appear at a point posite the attachment of the original gro This may occur especially in long-standing c in which the vascular polyp is located at the ju ture of the anterior and middle thirds of the v cord.

DIAGNOSIS

The diagnosis of vocal polyp is made by m of the laryngeal mirror. Direct laryngos is rarely necessary. A growth in the subgl region covered by an overhanging epiglottis require that procedure. Even when so situ a vascular polyp can be detected with the lar geal mirror if the epiglottis is anesthetized pulled forward. In all cases in which mi examination does not afford a clear view of laryngeal lesion direct laryngoscopy should performed.

Once the diagnosis is made, the polyp sh be removed if the normal function of the lar is to be restored. Recurrence is rare, but it is unusual for a similar growth to occur in ano section of the same vocal cord or on the oppo side. Six of the patients in this series retur with vascular polyps elsewhere on their v cords after periods varying from eight mo to sixteen years following the removal of original growths. Three of the patients had a previous similar growth removed. One ha

PROGNOSIS

The question is often asked: "What will h pen if the growth is not removed?" It is tremely doubtful whether this type of polyp undergoes cancerous change. Jackson was the opinion that noncancerous growths may come cancerous. Except for papilloma in ad this is not in accord with my experience. M laryngologists have stated that noncancer growths do not become cancerous. As time pa the polyp will become more fibrous and firm it will continue to cause hoarseness, vocal fati and distress, but it will not endanger the li its host. Because of the discomfort, annoy and embarrassment which usually accomp laryngeal polyp, removal should be recommen

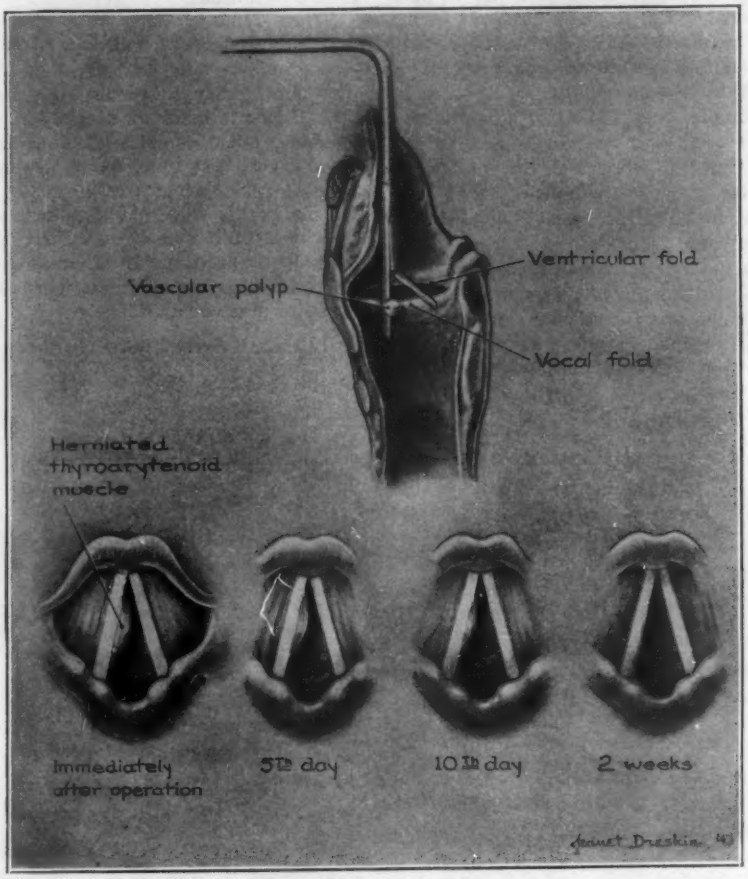
TREATMENT

emoval is the only treatment for vascular p of the vocal cord. Complete removal with- injury to surrounding healthy tissue is the ctive of the laryngologist. Local applica- s are of no value and may prove harmful; will vocal rest cause a vascular polyp to dis- ar, no matter how small it may be. Thyrot- or any other form of external operation is r necessary.

emoval of the growth is accomplished by ns of indirect or direct laryngoscopy and the quire the use of a complete operating room but can be performed in a cubicle or any suitable space set apart for it. 4. Removal of a tumor of the vocal cord by indirect laryngoscopy is a simple procedure when compared with direct laryngoscopy.

The indirect removal is accomplished as follows:

The patient and the operator are seated as for laryngeal examination. Attention is paid to the proper placement of the light behind the patient and the height of the patient's chair in relation to that of the specialist's stool. After cocainization of the larynx, the patient is



ig. 8.—Upper part: Removal of vascular polyp by indirect laryngoscopy. The forceps is about to grasp growth. Lower part: Mirror image of the larynx after complete denudation of the edge of the right vocal following removal of a vascular polyp. Healing was complete in two weeks.

of suitable forceps. Indirect laryngoscopy is erred for the following reasons. 1. The r equipment required is little more than that lly employed for the average laryngeal ex- nation: A patient's chair, an examiner's , a light which is reflected by the head mir- a laryngeal mirror and a special forceps are essentials. 2. The patient is in the sitting tion, completely at ease, and is not lying n, restrained by one or more assistants; no tional personnel is required for the removal e tumor. 3. Indirect removal does not re- asked to hold his tongue forward with the first two fingers of his right hand, using a piece of gauze for this purpose.

The larynx is brought into view by means of a mirror held in the laryngologist's left hand. A curved laryngeal forceps with a Frankel tip is held in the right hand. The forceps consists of a universal handle, to which is attached a curved laryngeal carrier. To the distal end of the carrier a Frankel tip is attached. The tip has two ovoid cups, the cutting edges of which are approximated when the handle is closed and separated when the handle is open.

The forceps are introduced with the cups approximated and guided into the larynx so that the tip rests on the neoplasm. The handle is then opened, releasing

the jaws of the tip so that they cover the growth. When the growth is on the edge of the cord, the flattened side of the opened tip is pressed against the cord, then closed, trapping the growth (upper part of fig. 8). The operator makes certain that the growth, and only the growth, is within the grasp of the forceps. When the growth is attached to the upper surface of the vocal cord, the technic for its removal is the same except that the jaws of the forceps are made to slide along the surface of the cord before the neoplasm is grasped.

After the forceps have been closed, a light twist of the handle removes the growth on a line with the edge or surface of the cord, and leaves in its place a superficially denuded area. The size of the denuded area usually corresponds to the extent of the surface previously occupied by the attachment of the growth.

A pedunculated growth which has a tendency to drop down under the vocal cord is best grasped during phonation, when the expiratory effort forces the growth on the vocal cord and fixes it there long enough for the operator to close his forceps on it.

Partial or even complete denudation of a vocal cord may occur when the growth is extremely firm or the forceps blades are dull or when the forceps grasps membrane beyond the growth. Although this is considered an accident, it is of no special significance, since even after complete denudation of a vocal cord epithelization occurs at the end of two weeks. In a woman 30 years of age the mucous membrane of the entire edge of the right vocal cord was removed together with a small vascular polyp. As a result of the complete stripping of the cord, the thyroaryten muscle herniated into the glottis, causing a large crescentic bulge. Daily examination revealed g diminution in the size of the protruding mass epithelium grew in anteriorly and posteriorly, the muscle back into place. The vocal cord ap normal at the end of two weeks (lower part of f

After-Care.—The postoperative care and tre of the patient are simple. The superficial wound by extirpation of the neoplasm is rapidly epitheli that the vocal cord appears normal within a few The greater the denudation, the longer the ti quired for healing. Occasionally a vocal co traumatized during removal of the polyp. As a the vocal cord becomes swollen and thickened, ing a week or ten days for complete resolution return to normal.

When removal of a vascular polyp leaves a small superficial denuded area, epithelization within twenty-four to forty-eight hours. It is fore unnecessary to require the patient who has operated on to maintain silence for periods of two or more, since complete healing occurs within days. It is best to examine the healing process so as to decide accurately when the patient may r the use of his voice. The patient should be instr to whisper for two or three days before returni normal vocalization. In all cases the patient shou warned against those things which constitute misu abuse of the voice.

136 East Sixty-Fourth Street.

ACUTE TONSILLITIS

TREATMENT BY RECTAL ADMINISTRATION OF A NEW BISMUTH COMPOUND

JOSEPH S. STOVIN, M.D.
NEW YORK

e of the commonest infections with which hysician has to deal is acute tonsillitis. Its om complex of fever, general malaise, sore , dysphagia and tenderness of the submaxil- ymph nodes is so well known that more d description is unnecessary.

ute tonsillitis usually lasts for a week to ten and is considered a self-limited disease ex- n the few patients in whom complications, as peritonsillar abscess or acute nephritis, . However, the patients are so uncomfort- nd the prostration so great that any ther- irected toward amelioration of the symp- and especially toward shortening the ion of the illness is a welcome addition to herapeutic armamentarium.

icylates and other antipyretic drugs have generally employed, but, while this medica- makes the patient more comfortable, it must nsidered as nothing more than symptomatic py; it certainly does not shorten the disease e incapacity resulting therefrom.

e bacteriology of acute tonsillitis is not defi- established. Beta hemolytic streptococci found most frequently, but staphylococci, mococci, Streptococcus viridans and Vin- organisms are also sometimes present.

cause of the frequent association of strepto- with the disease, the sulfonamide com- ds have come into prominent use. However, have been disappointing. In a study of 67 of streptococcic sore throat, Rhoads[1] treated atients with sulfanilamide and used 36 as ols. He concluded that the sulfanilamide not reduce the severity of the symptoms, en the period of incapacity, lessen the inci- e of complications or shorten the duration of arrier state. Moreover, in half of the patients hom sulfanilamide was administered, toxic festations other than the usual cyanosis red. He concluded, therefore, that sulf- mide should not be given routinely to the ge patient with uncomplicated tonsillitis or yngitis due to hemolytic streptococci.

In 1934, Monteiro,[2] of Brazil, published a paper advocating injection of a bismuth compound in cases of infection of the oral cavity with Vincent's organisms. He[3] followed this up in 1941 by endorsing the same therapy for catarrhal, follicular and parenchymatous types of tonsillitis. Subsequently there appeared many enthusiastic reports concerning the use of bismuth compounds in the treatment of tonsillitis. Some authors advocated use of a soluble bismuth compound, others use of the insoluble suspension in oil, but all corroborated Monteiro's original claim that with the injection of bismuth compounds the pain, temperature and malaise rapidly subside and the exudate and swelling of the lymph nodes promptly disappear. It has been generally agreed that the use of bismuth compounds reduces the duration of tonsillitis to twenty-four to forty-eight hours, instead of six to eight days.

Most of these reports have appeared in foreign publications. It seems that physicians in this country have been diffident about giving this relatively simple therapeutic procedure a trial.

Recently Lewis[4] published a series of papers on the causal relationship of Vincent's organisms to acute and chronic tonsillitis. Clinically, as well as by a study of smears, he determined that fusiform bacilli and spirochetes play an important etiologic role in tonsillar infections. He administered a bismuth compound (bismuth subsalicylate in oil) intramuscularly and found it a "highly effective agent in the treatment of most types of acute tonsillitis."

Just how the bismuth acts is not thoroughly understood. Pescetti[5] carried out histologic studies in 8 cases to determine the mechanics of

Rhoads, P. S., and Afremow, M. L.: Sulfanil- in Treatment of Sore Throat Due to Hemolytic tococci with Controls, J. A. M. A. **114**:942-943 rch 19) 1940.

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3. Monteiro, A.: Modern Treatment of Acute Tonsillitis by Injection of Bismuth Compounds, Arch. Otolaryng. **34**:719 (Oct.) 1941.

4. Lewis, J. F.: Fusospirochetal Organisms and Tonsillitis: Bismuth Therapy of Tonsillitis, Arch. Otolaryng. **35**:587 (April) 1942.

5. Pescetti, V.: Bismuth Therapy in Acute Tonsillitis: Experimental Demonstration of the Rapid Invasion of Tonsillar Tissues by Bismuth Salts, Arch. ital. di otol. **51**:607 (Dec.) 1939.

the curative action of bismuth. He injected a bismuth compound intramuscularly some hours before tonsillectomy was performed. He found that if the tonsils were removed within eight or nine hours after the injection, bismuth could be demonstrated microscopically in the tonsil tissue. After twenty-four hours, the tonsils were infiltrated with the bismuth.

Perhaps the chief reason that up to now bismuth compounds have not been in more general use in the treatment of tonsillitis is that they have necessitated intramuscular injection which is more or less painful and causes some induration and may give rise to abscess formation.

There is now a new method of administering bismuth which overcomes all the handicaps mentioned. It concerns a new bismuth compound (a salt of heptadienecarboxylic acid) which is readily absorbed when placed in the rectum in the form of a suppository.[6]

In an exhaustive biologic investigation of this preparation, Levenstein [7a] showed that in rabbits repeated administration caused no local irritation of the tissues of the anus and rectum and no general toxic reaction. Secondly,[7b] he demonstrated the absorbability of the bismuth when administered rectally by showing that when a rabbit was given 45 mg. of bismuth in the suppository form of the bismuth salt of heptadienecarboxylic acid in cocoa butter, there resulted an excretory level in the urine of 0.02 to 0.5 mg. of bismuth daily beginning within the first twenty-four hours after administration. This compares favorably with the urinary output of bismuth when a bismuth salt suspended in oil is given in comparable doses by intramuscular injection. He further proved its rapid absorption by demonstrating that after the insertion of even three suppositories no bismuth was found in the posterior 3 inches (7.5 cm.) of the rectum after seventy-two hours.

Observers do not know the exact pharmacologic action of bismuth. However, the fact remains that it has shown itself, in case after case, to be a valuable therapeutic agent and merits a high place in the therapeutic armamentarium against tonsillitis, especially since it is now available in a compound which is nontoxic, easily administered and rapidly absorbed.

I have administered these bismuth suppositories in a series of 40 cases from private practice and from the clinic for diseases of the ear and throat of the New York Polyclinic Hospital (10 of which I shall outline in detail) and have observed remarkable therapeutic results both in the speed and in the completeness of the amelioration of symptoms and signs.

REPORT OF CASES

CASE 1.—A woman of 32 years complained of a sore throat, headaches and pain in the back and thighs. When she was first seen her temperature was 102 F. and she suffered from general malaise. Her tonsils were edematous and covered with discrete patches of white exudate. A diagnosis of acute follicular tonsillitis was made and a bismuth suppository of the type described [6] was administered. That evening her temperature had dropped to 100 F. and the headache and backache were less severe. The next morning her temperature was 99 F. and all the general symptoms had disappeared. She was able to swallow more easily. The tonsils were smaller, although the exudate was still present. Another suppository was given, and by the next day all symptoms had disappeared.

CASE 2.—A woman of 26 years complained of a sore throat of two days' duration. When first examined she appeared to be acutely ill with general malaise and a temperature of 101.2 F. Swallowing was painful. The cervical lymph glands were large and tender. She had undergone tonsillectomy three years before. The lymphoid tissue on the pharynx as well as remnants of tonsillar tissue in each fossa were swollen and covered with a film of white exudate. A diagnosis of acute follicular tonsillitis and cervical adenitis was made and a bismuth suppository [6] administered. She was given a second one to insert twenty-four hours later. On the third day the general symptoms had subsided and she had returned to work. The membrane was still present, but the lymphoid tissue had shrunk. The glands were still enlarged but were no longer tender.

CASE 3.—A man complained of sore throat and difficulty in swallowing of one day's duration. Although he suffered from some malaise and headache, he was able to attend to his business. His temperature was 100 F. The tonsils were acutely swollen and covered with discrete white spots. A bismuth suppository [6] was given, and the next day the temperature was normal and all the general symptoms had disappeared. The tonsils were smaller and the exudate not as extensive as on the previous day. No further treatment was given.

CASE 4.—An 11 year old child had been ill for two days with a temperature range of 100 to 103 F. The chief complaints were sore throat, difficulty in swallowing, swollen cervical glands and pain in each ear. (The tonsils had been removed several years before.) The pharynx was acutely inflamed. There were several tonsillar tabs and some lymphoid tissue on the pharynx which were covered with a dirty white exudative membrane. The cervical lymph glands were enlarged and tender. Both drum membranes were normal in appearance. A diagnosis of acute tonsillitis and pharyngitis was made, and a children's size suppository of the type described [6] was administered. By the next day the general symptoms had subsided. Swallowing was easy and there was no pain in the ears. Another suppos-

6. The rectal suppositories used in this study were supplied by the manufacturer, Specific Pharmaceuticals, Inc., New York. Each suppository for an adult contains 0.135 Gm. of the bismuth salt of heptadienecarboxylic acid, corresponding to 0.045 Gm. (0.693 grain) of metallic bismuth incorporated in cocoa butter; the children's size contains one-half the adult dose.

7. Levenstein, I: (*a*) Leberco Laboratory, Biological Experiment R66, Jan. 9, 1942; (*b*) R231-2, Aug. 20, 1942.

iven, and by the following day all local and general oms had disappeared, although some weakness per-

E 5.—A boy 3 years old had been ill for two days first seen. Swallowing was difficult, and he com- d of pain in his left ear. The tonsils and the ids had been removed, and four months prior to esent illness I had performed secondary mastoidec- for acute mastoiditis on the left side. The tem- re now was 102.2 F. Examination revealed severe stion of the pharynx. The posterior tonsillar pil- were edematous. The tonsillar tabs were swollen overed with an exudative membrane. The cervical s were enlarged. The left drum membrane was sted, though all landmarks were visible. A chil- size bismuth suppository [6] was administered. On llowing day the temperature was 101.5 F. Gen- the child was greatly improved, although the local oms were still present. Another suppository was and on the next day the temperature was down Swallowing was normal. The pharynx was still ut not swollen. The exudate was not marked, and r was normal. No further treatment was given, he child soon recovered completely.

E 6.—A man complained of pain in the right side throat radiating to the ear and on swallowing, day's duration. His temperature was 99 F. The nx was strikingly congested. The right tonsil was en, bulging out toward the midline. It had a slight te on it. The soft palate also was slightly edema- My diagnosis was peritonsillitis on the right. A th suppository [6] was administered, and on the fol- g day all subjective symptoms had subsided. The was smaller in size (almost equal to the opposite and the peritonsillar swelling had been reduced. urther treatment was given. In this case the for- n of a peritonsillar abscess probably was aborted.

E 7.—A 7 year old boy had been ill for two days a temperature as high as 104 F. There was diffi- in talking and swallowing, and he complained of in the right ear. Examination revealed that he was ly ill with a temperature of 103.2 F. The tonsils greatly swollen, nearly meeting in the midline. were covered with numerous discrete white spots. cervical glands were large and tender. The right membrane was red in Shrapnell's membrane along andle of the hammer. All previous medication was ed and a children's size bismuth suppository [6] admin- d. By the following day the general symptoms subsiding. Swallowing was much easier, and there no complaint of the earache. The temperature was to 100.1 F. The tonsils were still swollen and ed with exudate, but this was less marked than e previous day. Another suppository was adminis- , and by the third day the child was much better. ever, his temperature continued varying between nd 102 F. for several days. Examination four days revealed a normal drum membrane. The tonsils still swollen, but the white spots were practically . The prolonged disturbance of temperature may been due to the accompanying adenitis, which had sted without causing any discomfort.

ASE 8.—A young man had been ill for one day with throat, pain and a burning sensation in the eyes, pain in the back and the legs. His temperature 100 F. The pharynx was congested. The tonsils been removed several years before, but secondary ths and pharyngeal lymphoid tissue were swollen and covered with small white patches. The conjunctivas were acutely inflamed, and pus exuded from the inner canthus of each eye. A bismuth suppository [6] was administered. The next day the temperature was normal and all general symptoms had subsided. The lymphoid tissue was still swollen, but there was no discomfort. Except for gargles with hot saline solution, no further treatment was recommended, and the patient recovered rapidly.

CASE 9.—A young woman had typical acute follicular tonsillitis with the customary malaise. On account of difficulty in swallowing she had not eaten for two days, and when first seen, on the third day, she was toxic and acutely ill. Following the administration of suppositories [6] for two consecutive days, the general symptoms quickly disappeared. The tonsils became smaller, and the white spots were barely visible.

CASE 10.—An acutely ill boy, 22 months old, was first seen on the second day of his illness. His temperature was 102 F., the pulse was rapid and the sensorium was dulled. Examination revealed a fetid breath, a heavily furred tongue, a generally engorged throat and edematous tonsils covered with an extensive pseudomembrane. His cervical glands were large, soft and tender. Material from the throat was cultured for Corynebacterium diphtheriae. (This culture proved to be negative.) One children's size suppository [6] was given. When he was seen twenty-four hours later, the picture had entirely changed. His temperature was down to 100.2 F., the pulse rate was 100, and he was bright and alert. As there was still considerable pharyngeal exudate and as the swelling of the cervical glands persisted, a second suppository was inserted. The next day, the temperature and the pulse rate were normal, the glands had decreased in size and the tonsillar exudate had almost completely disappeared. At this time there was some vomiting. This, however, rapidly subsided, and the patient made a quick and complete recovery.

SUMMARY

For many years, various observers have advocated the parenteral use of bismuth preparations in the treatment of acute tonsillitis. A new bismuth salt, that of heptadienecarboxylic acid, absorbable rectally, has recently been brought out in the form of a suppository. The use of this suppository eliminates the unpleasantness of intramuscular injection.

From a series of 40 cases of acute tonsillitis the following conclusions can be drawn. The administration of the bismuth salt of heptadienecarboxylic acid in suppository form can be recommended for acute tonsillitis. It has also proved efficacious in a large group of patients in whom tonsillar tabs and granulation tissues persisting after tonsillectomy had become secondarily infected. It rapidly relieves the general symptoms and sore throat, usually within twenty-four to forty-eight hours. The follicular exudate and cervical adenopathy subside somewhat more slowly. The use of more than two suppositories is rarely indicated. No toxic manifestations of the drug have been noted.

151 Central Park West.

Case Reports

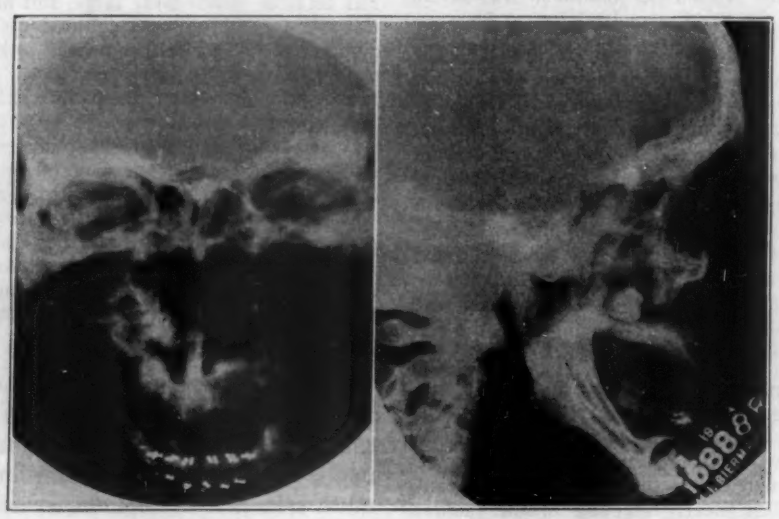
ABERRANT MOLAR TOOTH IN THE FLOOR OF THE NOSE

CATHARINE BIRCH, M.D., WASHINGTON, D. C.

A white man 39 years of age complained that he had been troubled with profuse purulent discharge from the right nasal passage for the past six months. His tonsils had been removed at the age of 5 years. At the age of 6 years treatment over a six month period was given for congenital syphilis on the basis of alleged dental malformation. At 8 years lupus vulgaris involving the nose and the shoulder was diagnosed, which healed ten years later. At 27 years a fracture of the nose resulted from an automobile accident. The Wassermann test of the blood had been repeatedly negative for the past ten years. All teeth were extracted at the age of 34.

Initial examination showed no sinus tenderness, but the right antrum was cloudy. The ethmoid cells were slightly cloudy on transillumination. The nasal septum was irregular but not obstructive. The turbinates were of normal size, with the mucosa slightly inflamed throughout. In the floor of the right side of the [illegible] surrounded by pus and impinging on the septum, [illegible] mass of ulcerated mucosa and what appeared [illegible] exposed bone 5 mm. in diameter. Roentgeno[illegible] showed a mass 1.5 cm. in diameter embedded [illegible] hard palate.

With the patient under nitrous oxide–ether anes[illegible] and after local application of cocaine and epine[illegible]



Roentgenograms showing a mass in the hard palate.

hydrochloride the mass was removed from the hard pa[illegible] by means of a slender mastoid gage introduced beneat[illegible] to dislodge it. The finger was inserted into the [illegible] terior choana of the right side. Because of the [illegible] of the object, it was necessary to remove it thro[illegible] the nasopharynx, and on delivery it was discovered [illegible] be a complete second molar tooth with decayed "ging[illegible] margin" and distorted roots. Recovery was uneven[illegible]

1835 Eye Street N. W.

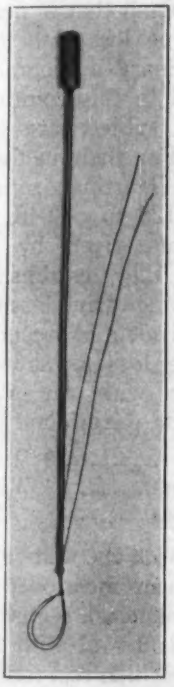
Clinical Notes; New Instruments and Technics

EASY LIGATION OF BLOOD VESSELS IN DEEP CAVITIES

A New Instrument and Method

CAPTAIN WAYNE Y. H. HO
MEDICAL CORPS, ARMY OF THE UNITED STATES

application of a ligature to control hemorrhage blood vessel in a deep cavity, such as the throat, est or the abdomen, is sometimes no easy matter surgeon. To facilitate easier ligation of bleeding of tissues in these relatively inaccessible regions, operations pertaining to the tonsils, the gallbladder,



g. 1.—Knot-tying instrument for easy ligation of vessels in deep cavities. It is shown threaded.

pleen, the kidneys or the lungs, instrumental meth- f tying knots are often resorted to.
e knot-tying instrument here presented is simple nstruction. It is inexpensive, practical and efficient.

rom the Children's Hospital, Los Angeles.

It is made of a long slender rod with two eyes on the nose at one end of it. The partition between the two eyes on the nose of this knot-tyer acts as the fulcrum, and all the force to be exerted in an easier knot-tying is being borne by this device, which eliminates simultaneously the possibility of tearing tissue free from the hemostat when ligating. This device is therefore unique for the purpose.

The technic is as follows: An ordinary slip noose is tied in the ligature, the free ends of which are threaded through the eyes on the nose of the knot-tyer. Then the noose is put around the bleeding point of tissue, already clamped in the usual manner with a hemostat. A slow even push of the knot-tyer coupled with a gentle

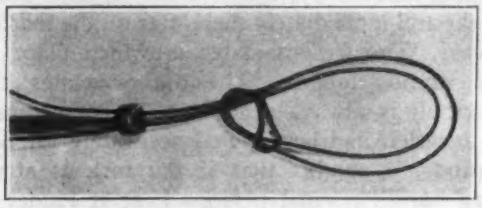


Fig. 2.—Method of making the noose and threading the nose of the instrument.

even pull of the two free threads contracts the noose firmly about the tissue. When tightly tied, the pull is relaxed and the knot-tyer is withdrawn, allowing the free threads to slip through the eyes of the instrument. The ligature is then cut free near the tissue.

For a large blood vessel, the question of the security of the slip noose ligature might be raised by some surgeons. However, this knot, secure as it is, can be further reenforced by first withdrawing one thread free from one eye of the knot-tyer and tying another knot with the other thread still hooked up with the nose of the instrument. Such a knot is as secure as any surgical knot.

Children's Hospital, Los Angeles.

Progress in Otolaryngology

Summaries of the Bibliographic Material Available in the Field of Otolaryngology

FUNCTIONAL EXAMINATION OF HEARING

ALFRED LEWY, M.D., AND S. L. SHAPIRO, M.D.
CHICAGO

War conditions continue to impose strong limitations on a report of progress in this field. Little continental European literature is available, and even the number of American and British contributions is less than in former years. Nevertheless, several worth while papers on hearing tests and hearing aids have appeared in the journals on hand, and these are summarized in our review.

Two suggested improvements in the use of tuning forks for testing bone conduction are given. Alexander [1] recommends a short cut for use in the "not so quiet clinic." After striking the fork moderately, the observer holds it before the ear and immediately thereafter on the mastoid process. The patient states in which place the fork sounds louder. The author states that the result is always practically as correct as if the duration had been timed accurately for both locations. He adds that if the fork is struck "very hard," normal persons hear it louder by bone conduction, but this phase is short and is probably due to stimulation of both hearing organs through the bone. This is indicated by the fact that in people with unilateral deafness the "loudness negative Rinne" phenomenon did not appear. In testing with the audiometer 50 or 60 decibels extra were necessary to carry the sound across the skull.

Netto [2] believes that for the Schwabach test the usual practice of placing the tuning fork on the vertex of the head is incorrect. He places the fork (A or a) on the mastoid process over the tip at five second intervals with a pause after each interval, timing the duration with a stopwatch. The result is expressed in a fraction of which the numerator is the number of seconds the fork was heard by the patient and the denominator the normal time as ascertained by examining a number of normal persons. Netto also uses the same method for "absolute bone conduction" after stopping up the external auditory canal. He feels that the vertex technic is faulty becau sound is perceived by both ears.

A method for constructing audiogram means of tuning forks is given by Dr. A Mazza and Nicolau Mazza.[3] They feel tuning forks are of great value in quali diagnosis and that all that is necessary in to measure hearing loss with them is to them to the ordinary audiogram. Papers viously published in this country by Roth Johnson on the subject are characteriz "rudimentary." For their method the Bra writers used the Hartman series of five running from c-128 to c-2048. Employing damping constants given by Fowler fr identical series, they establish by mean Fletcher's formula (hearing loss in decibels o Δ [t°-t]) the equivalent in seconds of the cri limits of hearing loss as shown in the audiogram made with the Western Ele audiometer. These were found to be as follo

Frequency	128	256	512	1024
Audiometer (decibels).....	94	105	115	120
Tuning fork (seconds).....	87	77	88	80

The vertical lines on the audiogram corresp ing to the frequencies mentioned are then div into equal segments, each segment corresponds to a five second interval with the exception the last segment, which represents the remai fraction; the usual normal line at the top of audiogram is assumed.

Certain criticisms or comments on the me should be stated. One, given by the aut themselves, is that the audiogram is limite the five frequencies mentioned; this can remedied by using additional forks. The aut apparently were content to accept Fowler's ibration for his series of similar forks. As i notably differ in quality and in rate of de ment even when they are of the same nu should be stressed that if accurate use of method is desired, calibration of the indivi forks by an accepted method is necessary. should also be stated that the figures for ext

1. Alexander, A. B.: The Binaural Phenomenon of Bone Conduction: A Tuning Fork Test, J. Laryng. & Otol. **57**:411, 1942.

2. Correa Netto, O.: The Best Technique for the Schwabach Test, An. Soc. mex. de oftal. y oto-rinolaring. **17**:9, 1942.

3. Mazza, A., and Mazza, N.: An Audiogram Means of Tuning Forks, Rev. brasil. de oto-laring. **10**:635, 1942.

ng loss calculated by the authors are y theoretic, as for some of the frequencies (e. g., c-2048) no tuning fork made at nt will emit a sound which lasts as long figure in seconds given in their calculations. ntandon [4] gives some details of a new meter built according to his specifications e Viennaphone Company of Vienna, Ger- . The instrument has a range of 25 to) cycles, is of the pitch range variety which es any tone to be tested, but is also gradu- n octaves from 32 to 16384 so that testing at octave intervals can be done if desired. intensity is graduated in decibels. The ment is now in use at Montandon's clinic eneva, Switzerland, and is described as h precision and easy operation. Montandon that it will be put on the market in the future.

omer [5] describes a method for testing pure hearing in small children. He claims that be used for the majority of intelligent en from 3 to 6 years of age who have ent usable hearing to respond to verbal ion. The equipment consists of a pure audiometer and several 8½ by 11 inch by 28 cm.) white cards on which have drawn or pasted colored pictures of objects the child normally associates with the ction of some sound and which are of st to a small child. Examples are pictures ains, airplanes, birds, Indians and lambs. best pictures are those which show an ani- "talking" to a person or to another animal; picture to a card is used. The picture is n to the child, and it is explained that one ng to let him hear the bird sing, the train le, and so on. The ear phone is then held s ear, and he is allowed to hear a tone priate to the object, e. g. a low-pitched such as c-256, with the picture of a train high-pitched one, such as c-8192, for a It is then explained that the child can the sound by putting his finger over the le of the engine or the mouth of the bird. s to touch the bird or the train as soon e sound is made and to lift the finger as ound is interrupted by the examiner. The r advises giving a partial test of not more four frequencies at a time because of the lty of keeping the child interested for more fifteen to twenty minutes.

comparison of mass testing with the speech graph audiometer and individual examination with a pure tone audiometer was made by Malis [6] in the course of a survey of hearing in the Los Angeles parochial schools. Tests were made on 2,567 children 5 to 16 years of age by means of a pure tone audiometer; around 20 per cent of the children were found to have impaired hearing. Malis points out that with the usual phonograph speech method of mass testing which has been in vogue hearing impairment has been found by various observers in 6 to 9 per cent of those tested. His conclusion is that the difference in the incidence found in his survey and that recorded by observers who used the phonograph audiometer is due to the fact that with speech tests high tone impairment is likely to escape detection.

In an article on the interpretation of hearing tests Kinney,[7] after reviewing the older methods, makes the point that while the audiometer furnishes a more exact means of studying impairment of hearing for pure tones, the degree of impairment indicated by the audiogram does not necessarily correspond to that for speech. Kinney cites patients from his own practice who showed little improvement for pure tones following inflation of the eustachian tubes but much better hearing for speech; on the other hand, he has seen patients whose hearing for speech was impaired but who showed no loss for pure tones. In connection with these observations he cites 2 patients each of whom had had a fenestration operation performed (by another otologist) whose improvement of hearing was much greater than could be demonstrated with the audiometer. His conclusions are:

1. An official standardization of the method of recording the hearing decibel loss should be adopted.

2. A person cannot hear pure tones binaurally any better than he can hear them in his better ear.

3. A person can hear the spoken voice better binaurally than he can hear it in his better ear if there is bilateral impairment.

4. The method of determining percentage loss of hearing adopted by the American Medical Association is far superior to any previous method. Because in the determination of the figures by the method of the American Medical Association more weight is given to the laboratory analysis than to clinical experience, the method is inaccurate in the following cases: (*a*) case of so-called otosclerosis in younger

Montandon, A.: A New Radio-Electric Audiom- Confinia neurol. **4**:366, 1942.

Bloomer, H. A.: A Simple Method for Testing earing of Small Children, Quart. J. Speech **28**: 942.

6. Malis, S.: Individual Audiogram Versus Mass-Testing as a Means of Detecting Hardness of Hearing, J. Internat. Coll. Surgeons **5**:498, 1942.

7. Kinney, C. E.: Interpretation of Hearing Tests, Laryngoscope **53**:223, 1943.

persons in whom the pure tone loss is much greater than the voice loss.

(*b*) Cases of unilateral nerve deafness in which the ability to perceive speech is practically nil while the patient still has some pure tone hearing.

5. While the ability to perceive and analyze pure tones may lie completely in the cochlea, the ability to perceive and analyze more complex tone patterns in the form of speech probably lies more centrally and has a bilateral innervation.

6. More accurate methods of estimating ability to perceive speech must be developed before true analysis of hearing loss can be accurately determined.

7. When these better methods are developed and a better understanding of the central connections of the eighth nucleus are obtained, some of the newer therapeutic methods for improving an impaired ear will be more readily evaluated.

A series of 1,365 school children, 8 to 14 years of age, was studied for two years by Loch [8] with regard to the presence and the permanency of tonal dips. The latter was defined as a depression for tones 512 to 4096 of at least 15 decibels or more below the average of the audiogram. For frequencies higher than 4096 cycles or lower than 512 the threshold for the next higher or lower tone had to be at least 20 decibels better than the dip frequency. Thresholds were determined for fourteen frequencies between 32 and 16,384 in a sound-proof room. It was found that dips occur about three times as often in boys as in girls and that the older children of both sexes showed more dips than the younger ones. About half of all dips were for the frequency of 4096 cycles. Industrial noise, stated by some writers to be a factor in similar studies made on adults, evidently is not a cause here, and the difference in incidence between males and females must be regarded as a true sex difference. Persistent dips were found most often for the frequency of 4096; about two fifths of all dips were proved to be permanent. During the period of observation the greater increase in dips for both sexes was at the frequency of 10321 cycles. Relationships were sought but not found between tonal dips in children and any of the following items: previous tonsillectomy and adenoidectomy; otitis media; common colds; the appearance of the membrana tympani; the condition of the nasopharynx and the season of the year.

Articles on hearing tests under military conditions continue to appear. Fry [9] observed that the present tests are not satisfactory in ex ing candidates for aviation and suggested what was needed was a method which indicate the suitability of a candidate fo duties he is to undertake, i. e., hearing would enable him to recognize auditory s whether speech or code. If there are gross of hearing for pure tones one can predict culty in understanding speech, but if this one ear only, the subject may be as good normal person in recognizing signals. A of hearing alone is not enough. Ability to i pret depends on intelligence. Another fac a background noise level of 120 phons. method involves placing the subject in a level noise field, wearing a standard s flying helmet; lists of words and sentence carried to him through a pair of telephone ceivers carried in his helmet. Gramop records are used. The relative levels of s and noise are changeable. A number of c dates can be examined at the same time. tions connected with the problem of signals

1. How far can training compensate for d of hearing as shown by the pure tone audiom

2. How much more difficult is it to tra person of defective hearing than a normal

3. What is the effect of monaural dea on the ability to receive signals?

4. What is the effect of frequency and in sity limitations inherent in the transmis system on a person with defective hearing?

Shilling and Everley [10] studied the effec exposure to gunfire in naval personnel respect to hearing. They divided their pati into two groups: The first gave no histor disease conducive to loss of hearing; the se gave a history that indicated a possible disposition either through disease or preexis acoustic trauma. They found that the first gr showed less hearing loss than group two. the latter there was indicated a loss of decibels below the average normal; the dea was of the perceptive type.

Loch [11] experimented with alterations o pressure in the middle ear to determine t effect on hearing. Examinations were made sound-proof room by means of the West Electric 1a audiometer. The results indic that both positive and negative pressure in middle ear, produced by the Valsalva me when of marked degree impair the thresh for frequencies from 32 to 1024 cycles by as m

8. Loch, W. E.: Incidence and Permanency of Tonal Dips in Children, Laryngoscope **53**:347, 1943.

9. Fry, D. B.: A Suggestion for a New Method of Testing Hearing in Aviation Candidates, J. Laryng. & Otol. **57**:11, 1942.

10. Shilling, C. W., and Everley, I. A.: Hea Loss Due to Exposure to Gunfire, U. S. Nav. M. B **40**:938, 1942.

11. Loch, W. E.: Effect of Experimentally Alte Air Pressure in the Middle Ear on Hearing Acuity Man, Ann. Otol., Rhin. & Laryng. **51**:995, 1942.

) to 30 decibels; those for frequencies from to 2896, 10 to 15 decibels; the thresholds 096 and 5793 cycles were not much affected; for 10000 to 13000 cycles showed more irment (30 to 35 decibels with negative ure; slight improvement on mild positive ure, lost when increased). With negative ure first high, then middle, then low tones impaired; no definite sequence was found positive pressure.

mpbell [12] lists six factors which may con-te to hearing loss in aviation personnel. e are (1) heredity, (2) age, (3) noise (most ent damage at 4096 double vibrations), (4) metric pressure (if it is not equalized through ustachian tube, aerotitis ensues) (5) disease, anoxia, which in his opinion is not a serious lem from the standpoint of hearing loss.

hilling and Everley [13] investigated hearing due to exposure to radio in submarine onnel. They found that permanent hearing is due to radio acoustic trauma. They decry ing receivers behind the ears for hearing bone conduction as useless from the stand-t of prophylaxis; the acoustic trauma con-es through bone and may even be greater g to the greater volume of sound necessarily by this method.

he same authors,[14] discussing auditory acuity ng submarine personnel, on the basis of 0 audiometric examinations give examples of e loss of hearing due to noise of Diesel nes. This damage is stated to be cumulative eventually permanent. It is also a fact that y men of long submarine experience have mal hearing.

he prevention of traumatic deafness is con-red all important by Wilson [15] since the con-on when due to acoustic trauma is not sus-ible to treatment. A review of the literature ws that methods of prophylaxis fall into nite channels. The most familiar include ices for plugging the external auditory canals, ying from the simple use of cotton with or hout the addition of some viscous substance different contrivances made of rubber or tic material; various pads and helmets are of value. All these means have a limited application in that they will not keep out more than 25 decibels and are objectionable for the reason that they cause some discomfort and inconvenience, particularly because they make conversation more difficult. Another approach—the elimination of noise at the source by substituting welding for riveting and similar procedures—is unquestionably the ideal approach to the problem, but its attainment is as yet far off. Wilson has taken advantage of the opportunity afforded for testing men in the Army both before and after they have been exposed to noise from rifle and pistol fire, to carry out a test first suggested by Peyser. This consists in taking two threshold values from an ear for a frequency of 256 cycles, one before and the second thirty seconds after the noise; an 80 decibel tone at that frequency is applied for five minutes. The object is to find those persons who have a definite lack of resistance to loud sounds as indicated by a rise in threshold of over 5 decibels after stimulation. A number of otologists since Peyser have conducted similar experiments but, as Wilson remarks, none have attempted to establish the validity of their results under actual industrial or war conditions. Wilson made Peyser's test on a series of 85 men, all of whom had been carefully studied for any factors which might obscure the results. Twenty-seven men showed abnormal fatigue and 58 did not; all 85 were later exposed to noise on the firing range over given periods under similar conditions. The audiometric studies subsequent to the exposure showed that of the 27 men who were considered susceptible 21 showed a hearing loss, while of the 58 insusceptible soldiers only 10 were affected. Wilson considers that his results clearly point to the value of using Peyser's or some similar method of screening out susceptible persons from occupations where noise is an important element.

An improved method of measuring the percentage of capacity for hearing speech from pure tone audiograms is presented by Fowler.[16] He observes that the main difficulty hitherto in devising an acceptable standard for this purpose lies in the fact that speech is made up of varying combinations of frequencies, consisting of broad and narrow frequency bands, all of which differ in intensity and importance under different conditions of production and reception. In recognizing speech one mentally interpolates certain frequencies which are not actually heard through acute perception of other frequency patterns which are normally associated with them, and this is an important phenomenon in the recog-

2. Campbell, P. A.: Effect of Flight upon Hear-J. Aviation Med. **13**:56, 1942.

3. Shilling, C. W., and Everley, I. A.: Auditory ity in Submarine Personnel: II. Hearing Loss Due Exposure to Radio Under Service Conditions, U. S. M. Bull. **40**:396, 1942.

4. Shilling, C. W., and Everley, I. A.: Auditory ity Among Submarine Personnel, U. S. Nav. M. **40**:27, 1942.

5. Wilson, W. H.: Prevention of Traumatic Deaf-: A Preliminary Report, Arch. Otolaryng. **37**:737 ne) 1943.

16. Fowler, E. P.: A Simple Method of Measuring Percentage of Capacity for Hearing Speech, Arch. Otolaryng. **36**:874 (Dec.) 1942.

nition of speech by people with reduced hearing. One can often miss hearing many articulations and even several words in a sentence without mistaking the meaning. The ability to do this is, of course, subject to great individual variations. Fowler believes that measuring the over-all intensity of speech does not make allowance for this phenomenon as well as for the fact that loud speech accentuates the low frequencies more than the high ones and that therefore such a method is unreliable for ascertaining the capacity to hear speech.

Fowler has taken four frequencies—512, 1024, 2048 and 4096—and by using the experiments of Fletcher and Steinberg, who determined the articulation loss after these and other frequencies were filtered out, as well as his own clinical experience with many audiograms demonstrating narrow "troughs," has constructed a table of values in terms of percentage for the stated frequencies. The ones below 512 and above 4096 cause so little loss that they can be ignored for practical purposes. Values given by Fowler for the frequencies mentioned are in order: 15, 30, 40 and 15, these numbers being used for multiplying the decibel loss at each of the four frequencies mentioned. Fowler feels that percentage loss of hearing for speech should always be based on the binaural hearing capacity for speech, as the poorer ear is of some assistance, especially in the localization of speech, and tests show that two ears hear better than one. In estimating capacity for hearing speech two other facts must be taken into account. The first one is that in nerve deafness the loss is not linear as in conduction deafness, owing to the recruitment phenomenon which enables persons with perception losses to hear sounds well over threshold nearly or just as loudly as normal persons. For losses over 40 decibels, however, the loss in clarity which accompanies recruitment causes so much "blurring" as to hinder hearing considerably; with losses over 45 or 50 decibels clarity is lost to such an extent in neural lesions that total deafness for speech is present although speech frequencies are still heard fairly well. Fowler has constructed a table for estimating the percentage of capacity for hearing speech, which takes all these factors into account; he feels that his method constitutes an over-all estimate, i. e., for far speech, near speech and speech heard at intermediate distances. In the discussion of Fowler's paper, Grove pointed out that neither Dr. Fowler's nor any other method for estimating loss of hearing for speech can be simple, one reason being that no single table can cover all classes of persons and all occupations. Macfarlan made a plea for the use of the phonograph audiometer with a decibel meter and a single intensity record, an apparatus w he stated has served him well for testing ability to hear speech.

Simulation of deafness is discussed by Gro who states that it may be conscious or un scious. The first variety of malingering is only one dealt with in this paper. If total ness is claimed, the patient must be obse over a period of time and caught off guard unexpected remarks or those that stir hi action, such as "you can go now" or "your p are open." People who are totally deaf a time develop keen tactile sense and notic nearby loud noise which a studied malinge will not. Other tests mentioned are the cochl palpebral and the Barth.

In feigned unilateral deafness malinger should be suspected if the patient makes movements of the spoken or whispered words parts thereof or fails to repeat words whispe at 20 cm. or voiced at 50 cm. with his good closed (this can be done normally with b ears closed); it should be suspected if he de hearing the tuning fork in the Weber test denies hearing it with the good ear closed also if on repeated tests varying results obtained. Malingering is definitely detected the Stenger test as described originally or modified by Wells; other tests are the Lombe the Ehrhardt, the Tschudi stethoscope (which is decried by the author), the Beck the Wotzilka lid movement test, the Marx, Nadoleczny, the Hummel, the Megoni and Doelger. Grove stresses the value of repeat audiograms made on different days in expos malingering. No mention is made of the T test for unilateral simulation or of the value the psychogalvanic reflex plus pulse and bl pressure curves (the so-called lie detector) total feigned deafness.

Currier[18] discusses the effect of office noi on audiometric results and notes that while the is a difference of opinion among otologists as the relative values of sound-proof and so-call quiet rooms, the latter term has not been defin Watson, for instance, has stated that the diff ence between a sound-proof room and a qu office is slight—at most about 5 decibels in t low tones—and concludes that the necessity f a sound-proof room has been overemphasized. number of papers dealing with hearing tests gi a "quiet room" as the site of the tests without an data as to the noise level, although it is wi known that 10 or even 5 decibels are of im portance, particularly in noting improvement

17. Grove, W. E.: Simulation of Deafness, Arch. Otol., Rhin. & Laryng. **52**:573, 1943.

18. Currier, W. D.: Office Noises and Their Effe on Audiometry, Arch. Otolaryng. **38**:49 (July) 1943

ing. The writer noted that existing informa- on average noise levels in different places, ding offices, varies considerably—a fact which loes not find surprising after watching the le of the noise meter in his studies. These with investigations in 15 test rooms in ear St. Louis. He found that the approxi- noise level was 35 decibels in the offices e otolaryngologists and less than 24 decibels heir testing rooms. Audiograms taken in latter were approximately 20 per cent less rate than the audiograms taken in the sound- fed room of the university, the tones below being the ones chiefly affected. He also mented on the fact that the technic used for iometric examinations varies greatly in dif- nt offices, making comparison of records from erent physicians quite difficult.

xperimental studies on a related subject— tory masking—are recorded in a paper by rak, Lindsay and Perlman.[19] The authors rved the influence of a masking tone on ring sensation and the acoustic middle ear x. Masking sounds were produced either ure tones or as noises by an audiometer as as by the Bárány noise apparatus and other ces. The subjects were rabbits whose tensor pani and stapedius muscles were exposed er light narcosis as well as a number of ents whose stapedius contractions could be rved through a perforation or a scar. The owing conclusions were made:

. The acoustic reflex of the tensor tympani scle in the rabbit is unchanged in the presence the typical noise background of a room or en a masking tone is sounded which is below threshold of the muscle.

. If the masking tone is above the threshold the tensor tympani muscle the acoustic con- tions of the muscle are superimposed on the scle and the threshold is raised.

. In the human subject masking causes a dip the hearing sensation without affecting the shold of the stapedius muscle.

. The absence of a masking effect on the ex is interpreted to indicate that masking inates outside the part of the acoustic system which hearing sensation and reflex response together. The two possibilities are: first, t the masking effect originates in the central vous system above the level of the reflex or rt from the fibers of the reflex arc; a second sibility is that the masking effect originates a different part of the cochlea.

5. The experiments indicate that the mechanical stimulation of the cochlea is undiminished in the presence of a masking tone.

6. As both the reflex contractions and the cochlear potentials are unchanged, the origin of masking is most likely not in the peripheral sense organ.

Of interest is a paper on the audiometric effects of voluntary contraction of the tensor tympani muscle by Smith.[20] Politzer and others have commented that contractions of this muscle diminished the hearing distance especially for the low tones. Dr. Smith reports his observations on a lieutenant who was able to contract his tensor tympani muscle voluntarily, this being visible by otoscopic inspection. The subject first discovered that he could perform this feat at about 8 or 9 years of age. The maneuver would produce a low-pitched roar in both ears and diminution in hearing sufficient to block out ordinary conversation; he could maintain a steady contraction of maximum intensity for twenty or thirty seconds; then the muscles would have to be rested for ten or fifteen seconds before they could be contracted again. Audiograms were made of his hearing under ordinary circumstances and during contraction of the tensor tympani muscle. The curve for the latter indicated a loss which was greatest at 128 double vibrations (the subjective roar corresponded to a tone just above 64 double vibrations) and gradually diminished up to 2048 double vibrations, which was unchanged; there was a further smaller diminution for tones above 2048, the greatest dip being at 4096 double vibrations. Captain Smith comments on the controversy regarding the effect of middle ear processes on the type of hearing loss and suggests that his observations may be of some value in the interpretations of certain audiometric records inasmuch as occlusion of the eustachian tube causes a retraction of the membrana tympani which places the aural conduction mechanism in a somewhat similar condition to that produced by contraction of the tensor tympani muscle.

Experiments on the stapedius muscle in relation to sound conduction were made by Wever and Bray.[21] They found that tension caused by a thread attached to the tendon of this muscle caused marked reduction in the electrical transmission for low tones, a smaller reduction for high tones, and for certain tones of middle range preliminary improvement followed by decline.

9. Kobrak, H. G.; Lindsay, J. R., and Perlman, B.: Experimental Observations on Auditory Mask- Laryngoscope **52**:870, 1942.

20. Smith, H. D.: Audiometric Effects of Voluntary Contraction of the Tensor Tympani Muscles, Arch. Otolaryng. **38**:369 (Oct.) 1943.

21. Wever, E. G., and Bray, C. W.: The Stapedius Muscle in Relation to Sound Conduction, J. Exper. Psychol. **31**:35, 1942.

They conclude that the stapedius muscle, like the tensor tympani, operates to protect the middle ear against stimulation. The pattern of distortion is also altered.

Perlman [22] presents a paper which attempts to revise the conventional ideas regarding conduction deafness in the light of experimental work done by European observers in recent years. The author observes that the old teaching that loss of hearing acuity for low frequencies means a conduction lesion and loss for high ones a nerve lesion is found to be incorrect from experience in audiometric examinations, tuning fork tests and loudness balance studies. One cannot predict the audiogram by objective examination of the ear because it is not known what the various components of the conducting apparatus contribute to the hearing curve and it is impossible to visualize more than a small portion of the structures in the middle ear that are concerned with sound conduction. Animal studies are handicapped by the lack of perfect methods for testing auditory function in animals, and the difficulty of applying acoustic physics to such studies further complicates the problem.

Perlman nevertheless concludes that a good deal of information on this subject can be obtained by observing the alterations produced on normal audiograms by certain specific lesions. To begin with the external auditory canals, a change here such as an almost complete occlusion by cerumen or by an osteoma, or conversely an abnormally wide canal, both being alterations which affect the resonance frequency of the external canal (normally about 3,000 cycles), does not, as would be expected, change the intensity of sounds reaching the tympanic membrane. Only when complete occlusion takes place does a form of conduction deafness occur, but here it is found that there is more interference with the propagation of high frequencies than with that of low ones.

There are three factors which control the response of any structure to sound waves: mass, tension or stiffness and resistance or damping. These factors will govern the kind of hearing impairment which will result from lesions of the conduction apparatus in man. The total mass of the drum and ossicles (which have a weight of only 0.05 Gm.) is small and is nullified still further by the highly efficient manner in which it is balanced around the axis of rotation. An increase in mass produced experimentally by injecting water or glycerin against the drum results in interference with high frequencies; similarly in cases of exudative catarrh (w fluid against the internal surface of the dr a loss for high frequencies is often seen, and or a large granuloma against the tymp membrane may give a similar hearing curve. the other hand, increase in the tension stiffness of the conducting mechanism with alteration in the mass or the damping sho according to the laws of physics, produce in ference with low tones. This condition prev when there is inequality of air pressure on two sides of the drum membrane and has b demonstrated to produce low tone impairm experimentally by Van Dishoeck, Fowler, We and others; clinically it is seen in the type deafness noted following the air pressure chan which occur in flying. Large losses in the dr membrane or the ossicles, affecting as they both the mass and the stiffness of the conduct mechanism, result in hearing loss over the en acoustic spectrum; ossicular defects (includ loss of continuity) are more important than ho drum defects, and separation of the incus fr the stapes is worse than loss of the mall Increased stiffness, which is typically manifes in stapedial ankylosis, produces low tone de ness, at least at first. Even in the absence o conducting mechanism, if the round and oval w dows are immobilized by connective tissue, use hearing for air-conducted sound is retained. T conducting mechanism is also affected by a condition which alters its damping ability, i its property of coming to rest quickly after t system has been set in vibration. An increa in the degree of resistance or damping of the co duction apparatus will cause an impairment the response to high tones. It must be tak into account, finally, that different frequenc vary greatly in their importance for hear speech, the vibrations between 1000 and 30 being of greatest value, and that intelligen occupation and other factors influence a perso ability to hear, so that one cannot predict t degree of handicap in any given case by looki at a threshold curve alone.

The interpretation of audiograms and t fitting of hearing aids formed the subject of address before the Royal Society of Medici by Fowler.[23] He stressed the importance the audiogram in general and its absolute nec sity for the prescribing of hearing aids. T necessity for a uniform technic in recording and bone conduction by the use of the symb familiarized by Fowler and the fact that

22. Perlman, H. B.: Lesions of the Conduction Apparatus, Arch. Otolaryng. **37**:680 (May) 1943.

23. Fowler, E. P., Jr.: Audiogram Interpreta and the Fitting of Hearing Aids, Proc. Roy. Soc. M **36**:391, 1943.

lard zero level of average normal hearing now been established by the Council of siotherapy were emphasized. A worth while sion in Fowler's paper consists of Swedish German audiograms as well as a comparison ese with the American type—a matter which ld be of interest to those American readers are unfamiliar with foreign audiograms.

mphasis is laid on the necessity for frequent ration of the audiometers as well as on the rtance of ascertaining the noise level in the g room. Fowler asserts that 15 to 20 els of noise are eliminated from the ear of patient by the use of the latest receiver, at noisy surroundings may lead to inaccurate ts and conclusions. He again stresses the that the presence of noise results in raising intensity of the examiner's voice during h and whisper tests. Rather than use a ant masking noise, as has been suggested, ler prefers to employ a sound level meter h records the intensity level of the exami- voice. By watching this apparatus, a pic- of which is included in the article, the r can learn to control his voice so as to it at an average level of intensity nearly f the time.

wler's latest method of estimating the nt's capacity to hear speech from pure tone grams (mentioned previously in this re-) is given in more detail as follows:

	500	1,000	2,000	3,000	4,000	8,000
	d. v.	d. v.	d. v.	d. v.	d. v.	d. v.
	15%	25%	30%	15%	10%	2%

umber of audiograms are included to show this method of weighting frequencies ishes an adequate explanation for the differ- in the capacity to hear speech exhibited by ons with hearing losses which at first glance quite similar. Fowler also explains the role cruitment of loudness in patients with partial e deafness in accounting for seeming dis- ancies in the ability to hear speech.

hen hearing aids are prescribed, certain facts be borne in mind. Ordinary speech in a room reaches the ear at a level varying 30 to 60 decibels, or an average of 45 els; below the 30 decibel level the inter- ation of speech becomes inadequate. For uction deafness hearing aids work well be- e, although considerable amplification may equired, interfering factors do not play as a role as in nerve deafness with which deli- adjustment for distance and voice must be e because of the recruitment factor. Fowler ves that air conduction hearing aids of the type are more efficient than the bone conduction type; that no patient with deafness on the average is less than 30 decibels above normal for the speech area and hardly any patient with a defect of over 85 decibels will be helped by a hearing aid; also that lip reading is generally best recommended ahead of a hearing aid, as few patients will bother to learn lip reading after they have acquired a hearing aid. A number of useful practical hints on the subject are also given.

In the discussion it was noticeable that British otologists were rather skeptical about accepting Dr. Fowler's dictum about the accuracy of the pure tone audiogram as a guide to the patient's ability to hear speech; nearly all of those who commented felt that speech tests, although imperfect, were essential. A good part of the discussion centered about modification of speech tests for examinations of military and especially air force personnel so as not to eliminate men who may have some impairment but are still fit for duty.

A paper on the hearing aid from the patient's point of view by Hughson and Thompson [24] states that the comparison, so often made, between fitting eye glasses and fitting hearing aids is inappropriate, as the first is an ancient art in which great skill has been achieved while the second is still in its infancy. Furthermore, there is an essential difference between the two procedures because in the vast majority of instances the eye accommodates readily for sudden extremes of light and darkness while the ear does not adjust itself well to sudden amplifications of sound such as occur when a hearing aid is worn over a long-deafened ear.

The use of a hearing aid is nevertheless the only form of therapy available for immediate help of the hard of hearing patient. Because the proper fitting of this device is a complicated problem, involving two equally important factors—the human ear with its concomitant psychologic aspects and the electrical device itself—the authors considered it worth while to question some 136 patients who had been advised to purchase hearing aids on advice given in the Otological Research Laboratory of the Abingdon Memorial Hospital.

For testing their patients the authors used a method previously reported. This employs the lowest measured intensity of spoken voice which can be heard as intelligible speech. A series of spoken sentences were delivered to the unaided ear at loudness levels controlled in 1 decibel steps.

24. Hughson, W., and Thompson, E.: The Hearing Aid from the Patient's Point of View, Arch. Otolaryng. **38**:252 (Sept.) 1943.

These tests in addition to pure tone audiograms were made with calibrated equipment in a sound-proof room. From a representative selection of different hearing aids, each patient was then tested in the same manner with at least the known characteristic response curve which is deemed suitable for the loss as shown by the audiogram.

Responses to a series of questions put by the authors were obtained from 97 persons who have been wearing hearing aids over periods varying from two months to twenty years. Seventy per cent of these patients declared themselves satisfied with their instrument. Of the group fitted by actual measurement of hearing gain, 80 per cent were satisfied. Ten different instruments and several different models of each aid were used by this group of patients. Of the vacuum tube aids, no single make is outstanding in its performance either by air or by bone conduction so far as the replies to this questionnaire indicate. The function of fitting hearing aids belongs, in the authors' opinion, to the otologist both because the psychologic makeup of the deaf person is an important factor and because fitting hearing aids is a form of therapy.

A paper on the acoustic properties of the external canal of the ear and of the middle ear with special reference to the fitting of hearing aids is contributed by Grossman.[25] The author believes that the average ear mold weakens the high frequency partial tones of speech, while on the other hand if the receiver of a hearing aid is connected with a flat ear piece these tones are perceived more clearly; patients suffering from a perceptive hearing loss hear better with a flat ear piece than with an individual ear mold. It is well known that the resonance imparted to speech by the external auditory canal depends on the length of the canal, its cross-sectional area and the impedance of the drum. The insertion of a channel in an acoustic conduit, either expanding or constricting in nature, renders the conduit frequency selective, provided that the cross section of the conduit is small compared with the wavelength. Grossman made experiments with different types of molds, all having the same length of 22 mm. but widths of 1.5 mm., 3 mm. and 5 mm., to find out how they influence the transmission of sound from the receiver of a hearing aid to the drum. His conclusions were as follows: The individual ear mold of present design influences the transmission of sound from the receiver to the ear by attenuating higher partial frequencies with resultant distortion. The design of mold for middle ear impairment sh be different from that for perceptive hearing The mold for the former disability should a long tip, and the diameter of the sound- veying canal should be not less than 3 mm. mold for perceptive hearing loss, on the hand, should have a short tip and the so conveying canal should be as large and as str as possible. A side branch orifice for the la preferable. In ankylosis of the stapes so transmitted from an air conduction rec acts as if transmitted to the ear by bone duction. Middle ear impairment of hearin not always of the obstructive type exclusi but produces symptoms which strongly sug pathologic nonlinear distortion of the so transducers in the ear.

In a report on the pellet type of artificial dr Lowy[26] states that this device when placed the stapes improves the hearing throughout hearing range, especially that for high to The mechanism appears to be composed of t effects: screening and increased stiffness of vibrating system. Bone conduction remains changed. Increased mass of the vibrating sys causes low tone improvement and (in the abse of screening) high tone loss.

The management of a hearing aid clinic described by Senturia, Silverman and Harris All tests are made in a quiet room. The sou of the sounds was an electric transcription Western Electric 2a audiometer and a mic phone, managed through a selecter switch, th through amplifiers to a dual speaker system co nected through an output selecter switch a with output volume indicator.

A complete history is first obtained and audiometric test is made with both bone and conduction receivers. The patient is seated a room simulating the reverberation charac istics of the ordinary living room and is placed that the ears and the hearing aid are 2 met from and at right angles to the source of sound. The pure tone and sentence tests then made and hearing measured for the unai ears; then they are repeated with the hear aid. The improvement with each aid is record and studied, and recommendations are ba on the patient's responses. The personnel the clinic includes a consulting otologist, train audiometrists, an acoustic engineer and an edu tor of the hard of hearing.

25. Grossman, F. M.: Acoustic Sound Filtration and Hearing Aids, Arch. Otolaryng. **38**:101 (Aug.) 1943.

26. Lowy, K.: Experiments on the Pellet Type Artificial Drum, J. Acoust. Soc. **13**:383, 1942.

27. Senturia, B. H.; Silverman, S. R., and Harris C. E.: A Hearing Aid Clinic, Ann. Otol., Rhin Laryng. **52**:131, 1943.

hree types of deafness are classified: con-
ion, mixed and perception. Thirteen makes
ids accepted by the Council on Physical
apy were tried, but in the results they are
gnated by code only. In conduction deafness
aximum of 30 decibels improvement at
ial frequencies was obtained. One aid gave
istently superior performance with a bone
uction unit. In mixed deafness a uniform
of 25 to 35 decibels was obtained. Varia-
of performance by the same instrument
ifferent cases was observed. In perception
ness a maximum amplification of 30 to 35
els restricted in most instances to the range
to 512 was given. The subjects with con-
ion deafness were most easily fitted, as the
occurred at those frequencies necessary for optimum intelligibility of speech. In mixed deafness the amplification could be spread over a wide frequency range giving better results than in perception deafness, in which the amplification was limited to a narrow band and extraneous noises dominated the lower portion of the spectrum, masking some tones necessary for intelligibility of speech. This problem is still a challenge. A gain of 10 decibels is often sufficient to make the difference between intelligibility in sentences. Perhaps it bridges a gap in the auditory pattern. Persons with extreme deafness cannot be fitted. A period of acoustic training is desirable after the acquisition of a hearing aid.

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Abstracts from Current Literature

Ear

THERAPY OF DEAFNESS: REPORT OF CASES. PART TWO. LOUIS GUGGENHEIM, Laryngoscope **53**:503 (Aug.) 1943.

Guggenheim states that over 50 per cent of the 18,000,000 deafened persons in the United States owe their affliction to tympanal disease resulting from obstruction of the eustachian tubes in early life. He points out that the usual "blind adenoidectomy" does not remove all the adenoid tissue and contributes toward future deafness. "Direct adenoidectomy" should be done instead.

The procedure is as follows: The Andy Love retractor is used to elevate the soft palate. A complete view of the nasopharynx is obtained. If the tubal orifices are visible, they should first be dilated with the Gerjoye dilator. Then the central mass of adenoid tissue should be removed with the La Force adenotome. After bleeding is controlled, the lateral adenoid tissue should be removed with the author's triangular forceps and curved scissors or with a nasal biting forceps. Finally the lymphoid tissue posterior to the posterior pillars should be removed. The instruments used for this are the triangular forceps and the curved scissors. Some three weeks later the first audiometric test should be done. Few recurrences of adenoid tissue have been seen after the use of this method as contrasted with the blind method. Under the title "Direct Adenoidectomy" this method was presented in detail in the ARCHIVES OF OTOLARYNGOLOGY (**33**:314-315 [Feb.] 1941).

The author explains the procedures followed in his clinic for deaf patients and tells how direct nasopharyngoscopy is performed by means of the Andy Love retractor. The view of the nasopharynx obtained is different from that obtained through the nasopharyngoscope. Most of the children examined, in whom tonsillectomy and adenoidectomy had been performed by the usual method, showed some lymphoid tissue about the eustachian tubes. Where there was no adenoid tissue the hearing was sometimes improved by tubal dilation. This improvement, he feels, was due to the initiation of resorption of tubotympanal mesenchyme.

In many children embryologic mesenchyme does not become resorbed. This tissue may change into fibrous tissue, which, in turn, causes conductive deafness. The failure of resorption may be a hereditary phenomenon and may explain why children of one family are afflicted with otitis media while those of another are not. The otitis media may be of a subclinical nature, with such slight symptoms that the child is never taken to a doctor.

The author presents photomicrographs of unresorbed mesenchyme in the middle ear, the mastoid antrum and the eustachian tubes. He emphasizes the fact that the unresorbed tissue is probably a great factor in the production of otitis media and deafness and hopes that a method will be found to influence its resorption. Anterior pituitary extract has been tried in human beings without success. Radium and roentgen therapy might help and should be tried.

HITSCHLER, Philadelphia.

MÉNIÈRE'S SYNDROME. J. HALLOCK MOORE, Laryngoscope **53**:519 (Aug.) 1943.

Moore gives a brief résumé of the modern the and treatment of Ménière's syndrome. He then re the physiology of the eustachian tube. The tube i only during deglutition, sneezing and yawning. C of the tube, with subsequent inequality of pressu the two sides of the drum membrane, causes a s secretion within the middle ear and hyperemia o drum head. This hyperemia extends to the tiss the middle ear and probably to the labyrinth. stapes is pushed in, causing increased pressure i labyrinth. Hearing is diminished under such cond Long-standing catarrhal otitis media might well c long-standing hyperemia of the labyrinth, as show Hallpike's autopsies. It would also explain the la other pathologic change at autopsy.

The treatment of Ménière's syndrome propose others, namely, a low intake of sodium and wa high intake of potassium and desensitization to h mine would benefit the condition by combating hyperemia. The cholinergic action of thiamine chl might explain its action.

Inflation of the eustachian tubes has relieved the s toms in many of the author's patients. The v was relieved in practically all of the 20 patients tr Allergic patients were treated by allergists, and eustachian tubes inflated occasionally, preventing return of symptoms. All nasal and endocrine dis were attended to.

The author feels that trouble with the eusta tubes may be the underlying cause of Ménière's drome and advises that thorough inflation plus ginage be performed before more elaborate thera attempted.

HITSCHLER, Philadelphia.

THERAPY OF DEAFNESS: REPORT OF CASES. T THREE. LOUIS GUGGENHEIM, Laryngoscope 5 (Sept.) 1943.

The author presents the results of treatment in dren under 13 years of age. The commonest cau deafness in children is tubal obstruction from ade and unresorbed mesenchyme. Generally speaking discovery and the treatment of such a condition to improved hearing in patients under the age o but probably not in those over that age, for perma tympanal fibrosis has taken place. The importanc discovering this condition early in life is there obvious.

Schools should require for admission of the chil examination by an otolaryngologist. The latter sh particularly test the hearing and examine the n pharynx by direct vision.

Crowe favors irradiation of recurrent adenoidal t while Guggenheim uses direct adenoidectomy.

A table is presented showing the results in 36 dren, all under 13 years of age, who had been treatment for impairment of hearing resulting tubal obstruction. The family history, the past hist the results of physical examination and the audiog are given. Depending on the case, the treatment sisted of any one or any combination of the follow tonsillectomy, direct adenoidectomy and tubal dila The average gain in hearing was approximately decibels for each ear.

HITSCHLER, Philadelphia.

Pharynx

ATMENT OF VINCENT'S ANGINA OF THE TONSIL. S. LINTON, J. A. M. A. **123**:341 (Oct. 9) 1943.

nton briefly comments on the therapy of Vincent's a with particular reference to the cases seen in armed forces. In addition to his discussion he nts a few case reports.

all the methods of treatment the following has adopted as the simplest and most practical: A Gm. sulfathiazole tablet should be dissolved on the ue every two hours during the day and a 1 Gm. t dissolved on the tongue every four hours during night; this plan is to be continued for two days, at h time the patient will either discontinue it volun- because symptoms have disappeared or continue little longer if necessary. If infection is present about the gum margins, it is advised that the thiazole tablet be moistened with a few drops of r to make a paste and this rubbed into the gum ins.

GORDON, Philadelphia.

APHARYNGEAL ABSCESS IN CHILDREN. JUAN CAROS OREGGIA, An. de oto-rino-laring. d. Uruguay 2:210, 1942.

reggia presents 2 interesting cases of parapharyn- abscess in children:

ASE 1.—A 3 year old boy stuck a glass tube into mouth forty-eight hours before admission. The tube e, and although several pieces were removed, a ment about 2 cm. long remained in the right para- illar region. On admission to the hospital he was emely toxic and pale and his temperature and pulse were elevated. The wound was explored in the of finding the fragment, but since this search was ccessful, the wound was packed with iodoform e. Five days later an extensive hemorrhage rred. It was controlled by tamponage, transfusion, se of coagulants and other measures. Ligation of carotid artery was advised, but the parents of the refused consent to this operation.

he child continued to be toxic and to have an eleva- of temperature, severe anemia and leukocytosis. days after the first hemorrhage, a second occurred h in a few seconds caused his death.

ASE 2.—A 19 month old infant was admitted to the ital with a high temperature and marked debility hree days' duration. The symptoms continued, and he twelfth day roentgen examination revealed a olidation of the upper lobe of the right lung. Two s after admission the patient was found to have y lesions in the mouth. These were both ulcerative necrotic and caused inflammatory swellings in the and in the floor of the mouth. These lesions pro- sed, causing marked necrosis of the left anterior r of the velum palatum. A culture from this region aled a pure growth of an anaerobic streptococcus. rvical gland was excised, but the pathologic report ed inflammatory adenitis. However, four days later child died, and a final diagnosis of typhoid fever made.

discussing these cervical infections, the author lays hasis on three common sites of involvement: (1) een the internal pterygoid and the styloglossus cles, (2) along the course of the styloglossus muscle nvade the floor of the mouth and (3) along the se of the stylopharyngeus muscle to infiltrate the ngeal operculum. In consequence one may observe r a submaxillary abscess, Ludwig's angina or ngeal obstruction. Extension of the suppurative ess along the large carotid vessels into the media- m can produce gangrene within the thorax, or pass- ing along the larynx it may produce suppurative bronchopneumonia. At other times striking arterial necrosis may occur with fatal hemorrhage (as in case 1). Other sequelae are sepsis, gangrenous bronchopneumonia and asphyxia.

The author advocates early surgical drainage, ligation of the carotid artery in the presence of threatened or actual hemorrhage, and tracheotomy if asphyxia is imminent.

PERSKY, Philadelphia.

Nose

SINUSITIS AND INFECTIONS SECONDARY TO THE COMMON COLD. F. M. TURNBULL, W. E. HAMILTON, E. SIMON and M. F. GEORGE JR., J. A. M. A. **123**: 536 (Oct. 30) 1943.

Following the work of Turnbull, who reported that use of a 5 per cent solution of the sodium salt of sulfathiazole brought relief from the symptoms of chronic sinusitis, the authors set out to investigate further. They found this solution unstable when exposed to light and air and that crystallization and discoloration were the rule.

They added sodium sulfite and found that the solution was then stable to air, light and heat. A vasoconstrictor dl-desoxyephedrine hydrochloride (discovered by Ogata in 1919 and like the original sulfanilamide a "sleeper" for twenty years), was found compatible with sodium sulfathiazole and actually formed a new sulfonamide drug, desoxyephedronium sulfathiazole.

Synergism developed so that only a small portion of the vasoconstrictive agent was necessary to achieve the hoped-for results.

Packs saturated with the substance were placed in the acutely swollen membranes of the nasal cavity and kept in situ for twenty to thirty minutes.

The clinical studies were applied over a wide range and in cases of acute, especially severe, disease, in which the mucosa was engorged. The patient was placed in the modified Proetz position and was given at five minute intervals instillations of 10 to 15 drops of this solution until the congestion of the tissues was sufficiently reduced to give relief from pressure.

Home treatment consisted of instillations of drops or sprayings at five minute intervals, usually two or three, until the deeper tissues of the nose were reached.

Acute pharyngitis and laryngitis were treated by spraying the nose and the throat and, in office treatments, the larynx and the upper part of the trachea.

Acute suppurative otitis media was treated by myringotomy and insertion of medicated tampons in patients treated in the office and by drops in the ear, the nose and the epipharynx when the treatment was given by the patient at home.

Chronic suppurative otitis media was treated by instillation of peroxide drops followed by insertion of tampons; home treatment consisted in the prescribed use of drops.

All of these measures were found safe and effective.

The authors emphasize that the methods outlined are not to be considered a cure-all, nor can they supplant indicated surgical treatment.

GORDON, Philadelphia.

Miscellaneous

A STUDY ON THE VALUE OF A MIXED BACTERIAL "ORAL COLD VACCINE." MORRIS SIEGEL, MARIAN G. RANDALL, MURIEL D. HECKER and MABEL REID, Am. J. M. Sc. **205**:687 (May) 1943.

The evidence outlined in this report indicates that the oral administration of a commercially prepared vac-

cine of mixed killed bacteria failed (1) to decrease the incidence of acute infections of the respiratory tract, (2) to reduce the severity or the duration of such infections and (3) to prevent secondary bacterial complications. A group of persons observed for purposes of control showed results remarkably similar to those in the persons receiving vaccines. Thus, both control and inoculated groups had exactly the same number of colds, and there was no significant difference between them in the duration of symptoms and in the number and the variety of complications following the colds. These observations strengthen those already made by various investigators that, contrary to fairly widespread medical and lay opinion, "cold vaccines" are of questionable value when used prophylactically against the common cold and its bacterial complications.

DAVISON, Danville, Pa.

THE RELATION OF CILIARY INSUFFICIENCY TO DEATH FROM ASTHMA AND OTHER RESPIRATORY DISEASES. A. C. HILDING, Ann. Otol., Rhin. & Laryng. **52**:5 (March) 1943.

Hilding discusses the changes found in the bronchial epithelium of patients who died of bronchial asthma, influenza and tracheobronchitis (1 case). In those with asthma the change consisted in a metamorphosis of the normal ciliated columnar cells into goblet or goblet-like cells. The ciliary mechanism was lost, and the characteristic viscid mucinous secretion accumulated in the air passages. Normally this secretion is carried up by ciliary action. In these patients the ciliary action was lost, the amount of secretion increased due to the change into goblet cells and the secretion remained attached over large areas within the cells which produced it; thus the mass was anchored to the wall. When the air passage had become sufficiently filled, the patient died of asphyxia. Hilding holds that the metamorphosis is the chief pathologic change and that death results directly from loss of ciliary action. In another group of asthmatic patients the cilia were also lost through a sloughing off of the surface of the bronchial epithelium. Chronic bronchitis with purulent secretion was prominent. The air passages were so filled with secretion that the patients died. Presumably the stasis and accumulation occur largely because of loss of ciliary action.

In the patients who died of tracheobronchitis the ciliated epithelium was entirely destroyed and the bronchi contained plugs of viscid secretion. Presumably this secretion could have been removed had ciliary action been normal.

In the influenza epidemic of 1918, many died with the lungs and air passages filled with a frothy thin liquid secretion. As nearly as could be determined the bronchiolar epithelium had been entirely destroyed and all the cilia with it. This type of secretion is not removed as rapidly by ciliary action—at least against gravity—as the more viscid types, but undoubtedly the loss of ciliary action was at least a factor in the accumulation of quantities of the secretion in the air passages.

In all of these groups, Hilding believes, the mechanical removal of secretion as a substitute for ciliary action is indicated. Aspiration either through a bronchoscope or a tracheotomy tube would be the method.

M. V. MILLER, Philadelphia.

PENTOTHAL SODIUM IN EAR, NOSE AND THROAT SURGERY. GEORGE J. THOMAS, Ann. Otol., Rhin. & Laryng. **52**:35 (March) 1943.

Pentothal sodium is a valuable adjunct to anesthesia. The compound has won a definite place in the surgical field. Among several anesthetic ag for intravenous injection, pentothal sodium was fou have the following advantages over other ag studied: (1) It is equally if not more powerful rapid in action; (2) twitching and jactitation are r (3) postanesthetic nausea and vomiting are less quent; (4) relaxation appears more satisfactory; recovery is more rapid.

A study of its action as regards human subjects sulted in the following observations: 1. It is a powe respiratory depressant, affecting the amplitude ra than the rate. 2. Laryngospasm, sneezing, coug and hiccups occasionally occur spontaneously or a result of some stimulation. Laryngospasm will variably cause alarming cyanosis, and if permitted continue, will cause cardiac embarrassment. These actions may be prevented by giving atropine su one-half to one hour before operation. 3. The bl sugar is slightly increased but returns to normal wi four hours after the administration of the drug. There is no hemolysis or change in blood clott 5. There is no change in the blood count, the dif ential count or the hemoglobin value. 6. There i drop in blood pressure of 8 to 40 mm., depending the rapidity of administration and the amount of d used. The hypotension parallels the depth of narc and is probably caused by reduction of the activity the sympathetic nervous system and by deprivation oxygen and not by toxic effect on the myocardi 7. The pulse rate increases by 8 to 30 beats a min depending on depth of narcosis and oxygen want. The electrocardiogram shows no deleterious effect the myocardium or the conductive system of the he 9. Pentothal sodium is detoxified in the liver, but metabolic activity does not appear to be impair 10. It does not affect the kidneys, and even advan renal inefficiency is not a contraindication to its u

Pentothal sodium is useful as a general anesthe and is ideal for surgical operations on the mast process, the sinuses, the nasal septum and the laryn it is useful in induction of anesthesia preliminary to t use of gas or ether; it is useful in supplementing loc or spinal analgesia, in combating any toxic effects a local anesthetic and in relieving the convulsive st that occur with drug poisoning, tetanus, eclampsia a ether anesthesia. It may be administered rectally intravenously; the former is ideal for inducing unc sciousness and averting physical shock while the pati is being transferred from his bed to the operating roo but the latter is the more practical.

The contraindications are as follows: Pentot sodium should not be used if there is marked physiolo or mechanical interference with respiration, if there an inflammatory condition of the neck complicated edema of the glottis or if there is a tumor of the re interfering with respiration. Children under 7 or unless robust, are poor subjects. In respiratory emba rassment due to cardiac decompensation, in bronchi tasis, in severe anemia and in shock it is contraindicate It should not be used preliminary to abdomi operations in which deep relaxation is important.

Preoperative sedation and the administration of atr pine are essential, as thereby hyperactivity of t parasympathetic system is controlled and salivary a mucous secretions are inhibited. This reduces suc complications as coughing, sneezing and laryngospas

The dosage must be adjusted to the individual patie as there is no general rule. Three cubic centimeters a 4 per cent solution is injected during ten to fifte seconds. As relaxation comes on more slowly th unconsciousness, a pause of seven seconds must follo the injection of each 2 or 3 cc. This is continued un

desired relaxation is secured. The air passage be patent. Oxygen must be administered with a machine, with a nasal adapter or with a 22 French ter (whistle tip) inserted for a distance of 5 inches cm.) through the nose and into the pharynx. venous injection of this agent should never be ed unless there is available oxygen, either alone n a gas machine. A "double setup" is described se when a supplemental solution, either a dextrose ion or an isotonic solution of sodium chloride is given during long operations. The supplemental ion is given continuously, while the solution of pen- l sodium is given intermittently.

r bronchoscopy, esophagoscopy, and operations on throat or the larynx, it is advised that before the othal sodium is given each piriform sinus be hed with 2 per cent tetracaine hydrochloride U. S. P. per cent cocaine. This is repeated once. The icator is held to the piriform sinus for one-half to minute each time. This is followed by instillation cc. of 1 per cent larocaine hydrochloride into the hea.

all patients anesthetized with pentothal sodium, gen should be administered continuously after the ation until they have reacted completely. Post- atively, opiates should be withheld, especially from patients, until they have reacted completely. If tion does not take place within two or three hours, xic dose has been administered. In this case the nt should be kept warm; oxygen should be admin- ed continuously; mucus should be aspirated from throat frequently; 1 cc. of picrotoxin U. S. P. should njected intravenously every twenty minutes until a t twitching of the facial muscles is noted, then intramuscularly every three hours until the patient ts. (Extreme caution must be used, as severe con- ions follow overdosage.) A solution of sucrose ld be given intravenously for diuresis and dehydra- of the brain and the lungs.

M. V. Miller, Philadelphia.

NCIPLES OF DIAGNOSIS AND TREATMENT OF ALLERGY AS RELATED TO OTOLARYNGOLOGY. FRENCH K. HANSEL, Laryngoscope **53**:260 (April) 1943.

llergy is increasing generally. This is because of easing contact with industrial and automobile dusts, ospheric molds and pollens, various chemical sub- ces, drugs and serums.

he diagnosis depends on a complete history and sical examination, together with cutaneous tests, logic examination of secretions, roentgen study of sinuses and bacteriologic investigation. Cytologic mination is emphasized.

here are several groups of allergic manifestations. se most interesting to the otolaryngologist are the ennial nasal and sinal allergy, allergy of the eye ear, hay fever, allergic bronchitis and bronchial ma. These are caused mostly by inhalants. Other ps of allergies are given.

t is well to emphasize that the ear may be involved various ways. There may be eczema and contact matitis of the auricle and canal. There may be iness. This deafness may be a result of swelling of eustachian tube or of involvement of the eighth ve (serum sensitivity). Ménière's disease may be result of allergic sensitivity in some patients.

he salivary glands may also be involved. A pain- swelling over the parotid area usually results. It be bilateral or unilateral. It is caused by a thick us plug obstructing Stenson's duct. The saliva con- s many eosinophils.

Before removing tonsils and adenoids for the relief of recurring head colds and bronchitis, one should be certain that the colds are not of an allergic nature. One should investigate chronic coughs. These may also be allergic and lead to asthma.

Only rarely does a food allergen cause allergy of the respiratory tract. Cutaneous tests are reliable for pollen sensitivity but often fail to reveal sensitivity to other inhalants (e. g., dust) or to foods.

For allergy due to an inhalant the treatment may be merely the avoidance of the allergen. Dust-proof pillow and mattress covers may be employed. Nonallergic cosmetics should be used. There should be no household pets. Sometimes a change of occupation or of residence is necessary. Of the inhalants outside of the pollens, dust is the most common offender.

The author describes a method of preparing extracts of dusts, and other antigens and gives charts showing cutaneous reactions to these antigens. He discusses the strength of solution used and the intervals of administration. He points out that most poor results are due to giving too large initial doses or to increasing the doses too rapidly. In regard to dust allergy the doses should be adjusted to the symptoms rather than to the cutaneous reactions.

Atmospheric pollen counts are important in the evaluation of hay fever. A technic for making these counts and a list of the important pollens are presented. In hay fever, cutaneous reactions are reliable. The scratch or cut method is satisfactory for diagnosis.

When treatment by preseasonal subcutaneous injections is not satisfactory, the intracutaneous method is sometimes beneficial. Coseasonal injections are often highly successful. As with the injections of dust, the initial dose should be small, and the subsequent doses should be regulated according to cutaneous reactions and symptoms. One should remember not to overstep the patient's tolerance. The optimum and not the maximum dose is to be preferred.

Hitschler, Philadelphia.

AURAL, ORAL AND LARYNGEAL TUBERCULOSIS. G. E. WILSON, New York State J. Med. **43**:680 (April 1) 1943.

Tuberculosis of the middle ear is secondary to pulmonary tuberculosis in adults and is seen in 5 per cent of all patients with this disease. Entrance is made via the blood, the lymph, the eustachian tube or a direct implant of the bacilli on surface contact. Symptoms are few; rarely there is pain. The time required to infect the middle ear is from one day to a few weeks. Single perforations are the rule. They may be ruptured tubercles(?). The bacilli may be found in a smear, by culture of material from the ear or by inoculation of guinea pigs with aural pus. There are round cell infiltration, giant cells, tubercles, granulations, necrosis or fibrosis. Syphilis must be differentiated. Tuberculous otitis is never in itself fatal. It is usually unilateral, with extreme deafness as an end result. Oral tuberculosis is not common, is secondary to involvement of the lungs and is "curable" if the pulmonary lesion is curable. Many of the oral involvements occur in men. The galvanocautery properly used effects healing. Laryngeal tuberculosis has decreased 50 per cent in the past ten to twelve years, perhaps because of collapse therapy, interruption of the phrenic nerve and thoracoplasty. Frequent examinations of the larynx should be made in all cases of tuberculosis of the lungs. Hoarseness may or may not be the first symptom, depending on where the lesion is situated. The voice may remain good in the presence of a bad epiglottic, arytenoid or "fold" involvement. One or both cords are most often

involved, and second in frequency is the posterior commissure, next the epiglottis and the arytenoids, rarely the ventricular bands. In unilateral lesions the greatest destruction is not always on the side of the pulmonary lesion. Tuberculosis of the larynx is usually slow, chronic, but may be fulminating. It is amenable to the use of the galvanocautery by the "indirect method."

VOORHEES, New York.

ACUTE TRACHEOBRONCHITIS COMPLICATED BY BRONCHIAL STENOSIS FOLLOWING THE INHALATION OF SULFUR DIOXIDE. JOSEPH GORDON, New York State J. Med. **43**:1054 (June 1) 1943.

Gordon cites Haggard, who pointed out that all irritants of the respiratory tract have the same toxicologic action. The relative solubility of gases in water has to do with the degree of pathologic reaction. A gas that is water hungry takes water out of the mucous membranes of the respiratory tract, and therefore its action is limited to the upper airways chiefly if the inhalation is not too long continued. However, a gas that takes up water slowly exerts its baleful influence in the lower portion of the tract and below the surface epithelium, even into the submucosa. In any case there are inflammatory edema and cellular exudate, followed by suppuration. Bronchiectasis may be an ultimate result. If there are ulcerations, stenosis from contacts across the lumen of the air tube may result.

The patient here reported on had a mild infection of the upper airways. He was accidentally exposed to fumes of sulfur dioxide. This resulted in tracheobronchitis. Treatment by means of a bronchoscope resulted in "clinical improvement." Some months later a second inhalation of fumes of the same gas took place from burning coke. The resulting tracheobronchitis produced partial stenosis of the main left bronchus.

VOORHEES, New York.

TREATMENT OF ASTHMA AND HAY FEVER. A CONFERENCE BY STAFF MEMBERS OF CORNELL UNIVERSITY MEDICAL COLLEGE AND OF THE NEW YORK HOSPITAL. HARRY GOLD, ROBERT A. COOKE, C. H. WHEELER and JANET TRAVELL, New York State J. Med. **43**:1224 (July 1) 1943.

The term "hay fever" is used to represent all types of nasal allergy. Pathologically there are two types: vasomotor (with acute and completely reversible reactions) and hyperplastic (with chronic and nonreversible changes in tissue). The treatment depends on avoidance of the cause, immunization and symptomatic medication. The patient may move to pollen-free areas, he may live in air-conditioned surroundings, he may eliminate certain household agents, e. g., pillows, mattress, chairs, pets, foods, insecticides. As for treatment of symptoms, little can be done with drugs. In general, local nasal treatment (sprays, cauterization and packs) are to be avoided, for they increase the irritability of the tissues and their action is only temporary. As yet there is insufficient evidence that deficiency of any vitamin or dyscrasia of any endocrine gland has much to do with allergy save as it affects the general health. Foods, contrary to popular belief, "rarely cause trouble," but air-borne substances are common factors. Adenoids, tonsils and teeth must be investigated, but the "type of infection" may be hard to determine. Autogenous vaccines are preferred to "stock" vaccines. Cultures from the submucosa are preferable to cultures from the surface. Cooke makes the following comments: 1. Rarely is surgical treatment indicated for a patient under 25 years of age. 2. Allergy due to inhalants or foods should not be treated with allergens before or after operation. 3. Surgical work must be "superlatively well done." 4. Operations on sinuses must remove infected tissue and secure "drainage"; turbinates must be conserved; resection of the septum is done chiefly to make the surgical approach to a sinus easier. 5. Sinuses are often operated on piecemeal, incompletely or too [illegible]. 6. Patients (for surgical treatment) must be selected with an eye to their physical condition. Not much can be expected when chronic pulmonary fibrosis is present.

Cooke speaks of asthma as (1) atopic, in which the cause is determinable by cutaneous tests, or (2) infective. The more difficult asthmatic patients are children who are continuously asthmatic but not completely disabled. If contact with an allergen is unavoidable, one must try to immunize the patient. This may require weeks, months or years and is vitiated by intercurrent infections of the respiratory tract, which often precede sinusitis and asthma. "Children up to 18 years need little attention to the sinuses if recurring infections can be controlled, and the early removal of tonsils and adenoids is one of the best methods of control." Infections of sinuses, especially those of the antrums, are the most frequent causes of infective asthma. Teeth are, of course, important foci, but are less important than other sources.

Summary by Dr. Walter Modell: The treatment of hay fever depends on whether the condition is of the vasomotor or of the hyperplastic type. If it is of the former type, avoid the cause, immunize, treat symptoms. If it is of the latter type, permanent changes of the tissues are likely, infection is probably present, and recourse must be had to the use of vaccines, surgical procedures or purely symptomatic treatment. Patients with urgent symptoms—bronchial obstruction, for example—may require removal of bronchial plugs by aspiration or administration of oxygen. Oxygen is given by catheter rather than by mask. Dehydration sometimes helps bronchial obstruction. A diet low in salt and sodium ash, together with sodium or poassium iodide internally, plus catharsis may afford relief. Severe reactions following the administration of allergens may require solution of epinephrine hydrochloride subcutaneously or, in rare cases, intravenously.

VOORHEES, New York.

A STUDY OF THE HEADACHES FOLLOWING DIAGNOSTIC SPINAL TAPS. HARRY ADLER, New York State J. Med. **43**:1328 (July 15) 1943.

Modern therapy calls for spinal tap in an increasing number of cases. This procedure is useful, but is frequently followed by severe headache and symptoms of an emotional nature. Adler studied 108 cases. The tap was performed at 2 p. m. as speedily as possible, with the patient erect. Rest in bed was avoided or delayed. This does not increase morbidity and lowers the percentage of headaches. Of the 108 patients, 14 had "moderate or severe reactions." "We feel that intracranial hypertension is the cause of the headaches. All these patients were under institutional care and observation for at least one year. Possibly the choroid plexus is influenced through the emotions. The fundi oculi shows edematous changes at times; consequently "we believe that headaches are due to an increased amount of fluid in the central nervous system."

Adler concludes as follows: "The sooner a patient goes to bed after a diagnostic spinal tap, the more he is apt to have a headache. The longer a patient is ambulant after a spinal tap, the less apt he is to have a headache and if one is present, it is likely to be mild. We believe there are two factors that predominate as to the causative factors of spinal headaches—namely, constitutional makeup and disturbed psychogenic influences."

VOORHEES, New York.

ITAL EMPHYSEMA SIMULATING CELLULITIS. C. L. C. O'MALLEY, Brit. J. Ophth. **27**:222 (May) 1943.

A case is described of severe proptosis, endangering eye, which followed a stab wound made by a foot- l player's finger. A slight hemorrhage from the eye l left nostril was followed by closure of the eye. ptosis began on the third day. The cornea became que and sloughed at the exposed area. There was rked chemosis, and the eyeball was stony hard. The ptoms were indistinguishable from those seen in ital cellulitis. An evacuation operation determined t the proptosis was due to gaseous accumulation in orbit. The eye, which looked a total loss, made an ost complete recovery.

To the author the most likely explanation is that the er made a valvelike opening in the orbital plate of ethmoid bone, which allowed air under pressure to er the orbit.

ZENTMAYER, Philadelphia. [ARCH. OPHTH.]

SPONSE OF PFEIFFER'S BACILLUS MENINGITIS TO SULPHAPYRIDINE. RAYMOND A. MOIR, Lancet **1**:556 (May 1) 1943.

Moir reports 4 cases of meningitis due to Haemo- lus influenzae. In only 2 of these 4 cases did recovery ur. He believes that the bacilli were too scanty in original smear of the cerebrospinal fluid for much dit to be attributed to the drug for the recovery in f the 2 cases. Fothergill and Sweet (*J. Pediat.* **2**: , 1933) reported only 1 spontaneous recovery in 78 es. The other recovery in Moir's cases (that of a month infant) he feels was a true cure, especially ce children under 1 year of age have not been known recover spontaneously.

In this series Moir noted that the temperature might p to normal only to rise again, whereas the pulse yed high until the temperature had fallen permanently. Hence the pulse is taken as a more reliable guide in prognosis than the temperature.

He recommends that lumbar puncture be done once for diagnosis, the procedure to be repeated only if restlessness and irritability become uncontrollable; otherwise, a second tap should not be done until the patient is clinically well.

Administration of sulfapyridine in doses of no less than 3 Gm. every twenty-four hours is advised, with continuation of the drug in reduced doses for two weeks after meningitic signs are gone.

McCARTER, Philadelphia. [ARCH. NEUROL. & PSYCHIAT.]

PNEUMOCOCCAL MENINGITIS: RECOVERY AFTER SULPHAPYRIDINE AND SERUM THERAPY. H. A. THOMAS, R. J. TWORT and C. P. WARREN, Lancet **1**:581 (May 8) 1943.

The authors report a case of pneumococcic meningitis following mastoiditis. The patient was first treated with sulfapyridine but in a few days was also given type-specific antipneumococcic serum, since he was not improving under treatment with the drug alone. There was rapid change for the better after the first 100,000 units (U. S. P.) of the serum, a change so dramatic that the authors suggest it was more than coincidence and probably indicates that the serum was the chief curative factor. Several days after convalescence had begun sciatic neuritis with foot drop developed on the right side. Intramuscular injection of the sulfapyridine is offered as the most likely cause of this complication. In the sixth month after the illness developed the patient suddenly had a focal epileptic attack, followed by hemiparesis and motor aphasia, from which he slowly recovered.

McCARTER, Philadelphia. [ARCH. NEUROL. & PSYCHIAT.]

Society Transactions

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY

SAMUEL J. PEARLMAN, M.D., *President*

WALTER H. THEOBALD, M.D., *Secretary*

Regular Monthly Meeting, Nov. 1, 1943

Anatomic Contributions of Vesalius to Otolaryngology. Presented by DR. IRVING S. CUTTER, Dean Emeritus, Northwestern University Medical School (by invitation).

Vitamins in Otolaryngology. Presented by DR. H. B. PERLMAN.

In looking for a relationship between clinical otolaryngology and vitamin physiology the soundest approach is to study the clinical and the pathologic picture of each known deficiency state in man. If there is no evidence of disease of the ear, nose or throat in a known deficiency state, it does not seem reasonable to expect benefit for the diseased ear, nose and throat from that particular vitamin.

While vitamin A deficiency in man has been widely studied, no characteristic otolaryngologic signs have been reported except occasionally metaplasia of the respiratory mucous membrane in children dying with such deficiency. Thiamine deficiency in man has been carefully studied. No otolaryngologic signs are reported.

Nicotinic acid deficiency does produce characteristic lesions of the mouth, the tongue and the esophagus along with the other classic findings. Redness, dryness, atrophy and small ulcers characterize the changes in the mucous membranes of interest to the otolaryngologist. These lesions respond to nicotinic acid therapy.

Riboflavin deficiency has also been carefully studied. Cheilitis and ragades at the corners of the mouth are characteristic findings. Before these lesions develop, burning of the mouth, tongue and eyes is noted. These respond to riboflavin therapy.

Ascorbic acid deficiency produces perifollicular hemorrhage and delayed healing of wounds only after prolonged complete depletion.

Vitamin D deficiency is not associated with any characteristic lesion of the ear, the nose or the throat.

Vitamin K deficiency causes a low prothrombin level in the blood and is important in patients with obstruction of the bile ducts and in the newborn.

In contrast to this limited relationship between known vitamin deficiency states in man and clinical otolaryngologic findings, many laboratory experiments on animals show suggestive relationships. An important example is the effect of vitamin A depletion of young animals on the length and the size of the internal auditory meatus. Marked lengthening and narrowing by new bone take place. This may lead to impaired function of the eighth nerve and changes in the end organs. No direct clinical importance can be attached to this experiment. Classic signs of vitamin A deficiency are practically unknown in this country, and in those countries where the disease is commonly reported this bony hyperplasia or functional impairment is not seen.

Until more is known about the physiology of vita and more widely accepted criteria are developed recognizing a deficiency state, further correlations otolaryngologic findings will be difficult.

DISCUSSION

DR. HOWARD C. BALLENGER: I had hoped Dr. P man would talk a little more about a possible vitam deficiency in chronic sinusitis. Perhaps he can elabo somewhat on that subject.

DR. THOMAS C. GALLOWAY: Dr. Perlman's car study merits close attention but I do think he is a l more discouraging than he might well be. It may be valid to say that if changes demonstrable in laboratory are established only with extreme deple of the vitamins, one may assume that there are no clinical changes with lesser deprivation. That partly depend on doubtful criteria. For instance, les about the mouth are taken as an index to ribofla deficiency. However, there are other less obvious f ings in riboflavin deficiency, such as vascularization the cornea, which can be picked up by the slit la This finding, associated with definite ocular discomf may be reversed by administration of the vitamin w relief of the patient's symptoms.

Similarly with vitamin A deficiency, certain cli states are suspect. A condition that is rather preva is itching eys, especially in dieting women or pati with disease of the gallbladder, whose food conta few sources of vitamin A. With the slit lamp th may be seen edema and changes in the corneal e thelium and, in extreme cases, keratomalacia. Th may be associated changes in the skin, including ke toses and even a tendency to formation of small ba cell carcinomas. This condition in some cases see to respond to administration of vitamin A.

I think it is important to expect definite findings the laboratory if one is going to have positive pr nouncements. On the other hand, one cannot ignore t factor of subclinical disturbances which may respond therapy even though this may lead to some exploita of the use of vitamins.

DR. A. R. HOLLENDER: The general consensus see to be that if a person is deficient in one vitamin he probably deficient in more than one. I happen to familiar with a certain study now being conducted the East. Large doses of vitamin A are being adm istered for certain types of deafness, particularly th with which tinnitus is associated. As much as 100,0 units is given daily, with results that seem highly e couraging, especially in controlling tinnitus. No rep has been made as yet, nor are any claims made co cerning this therapy. While vitamin A has been e ployed in cases of hearing defects, the large doses a advocated have not heretofore been suggested. The remains to be established a rationale for this treatm Another problem is the perfection of a product t dosage of which can be properly controlled.

While the essayist's theme is primarily vitamin I should like to bring up a point in connection w nicotinic acid. Most otolaryngologists are familiar w Atkinson's work on Ménière's disease. If Dr. Pe man's theory is correct, that the results from nicot

are purely transitory, Atkinson's suggestions con-ing nicotinic acid therapy should be discredited. ether the results with nicotinic acid are attributable egulaton of other existing vitamin deficiencies I do know. It is known that administration of thiamine rochloride, for instance, influences other vitamin ors. Not many years ago the Council on Pharmacy Chemistry of the American Medical Association e unfavorably of vitamin therapy. The situation changed. In a recent report the administration of tiple vitamins is no longer regarded as altogether tional if it appears that a deficiency of more than vitamin exists and it is impossible to determine ch are deficient.

R. IRVING S. CUTTER: I was tremendously impressed this paper. I am sure Dr. Perlman has followed work of certain men who are observing the catalytic on of certain vitamins, wherein they appear to rt synergistic effects. Particular fractions appear ossess the power to speed up reactions. This may lain in part clinical effects which may not coincide ctly with laboratory data.

R. WALTER THEOBALD: Dr. Perlman is to be com-ded for having taken a preparation which is handed at every drug store, given away in samples and sted over the radio and, with it, developed this ellent presentation. Parts of his paper are rather ouraging. Empirically otolaryngologists are all giving mins, and I had hoped to learn whether I could vitamins for degenerative nerve conditions of the I have tried large doses and have obtained what ought were favorable results in a few cases. Whether results were due to the vitamin, to psychologic in-nce or to God, I cannot say, but now and then good lts are obtained. I wish he could tell what effect mins have on the bone of the cochlea in the human g if any.

R. H. B. PERLMAN: Dr. Ballenger asked about the tion of vitamin A deficiency to sinusitis. There are orts of autopsies on children dying with xerophthal-. There is an occasional instance of metaplasia of respiratory mucous membrane from columnar ated to pseudostratified. I had occasion to talk with e of the men from South America who attended last meeting of the Academy about this problem. y see many classic deficiency states and often, ociated with the classic findings, there may be usitis or otitis media. I asked if they could recognize deficiency state by these findings alone and they that they could not. Remember further that vita-A deficiency as evidenced by xerophthalmia is prac-lly unknown in this country.

r. Galloway spoke of the vascularization of the nea as a sign of riboflavin deficiency. That is one he more recent attempts at early recognition of a iency state. Another method is that of determining much of a given vitamin is kept in the body after nown amount has been fed and how much is ex-ted. Criteria for recognizing a deficiency state are in process of formulation. Some are widely epted; others are open to criticism. Slit lamp find-s in the eye might be checked against some more ntitative method. The qualitative determination of min A in the plasma of a depleted animal shows rcut marked reduction over the controls. Chemical hods are probably least open to criticism. Studies correlation between biophotometer tests and the level vitamin A in the plasma have as yet given no con-ive results. As to the relation between the hor-nes and the vitamins little is known. Some of the studies in the otolaryngologic field that have been published entailed the use of both vitamins and hormones in a single patient. It was practically impossible to arrive at any conclusion as to what, if anything, was having an effect. Hormone deficiency states are also often hard to define.

Dr. Hollender mentioned the use of large amounts of vitamin A in the therapy of deafness. Little is known about the physiology or the pharmacology of massive doses of vitamins. Some vitamins are harmless in overdoses, usually being promptly excreted. Others may be harmful in massive doses. I am not sure what place nicotinic acid has in the treatment of Ménière's disease. Perhaps some acute episodes are being reversed by its use as a vasodilator.

Dr. Cutter mentioned the catalytic action of the vitamins. Information about the biochemical properties of vitamins has grown rapidly. The experimental literature is filled with such reports. Riboflavin is a widespread catalyst for cell oxidation. Synthesis of vitamins in the body, interaction of one vitamin with another and many other basic observations are being made. However, as clinicians, otolaryngologists must keep a proper perspective when attempting to find a new therapeutic approach.

Dr. Theobald asked about large doses of vitamins for nerve deafness. I do not know the rationale or the results of such treatment. As to the effect of vitamins on the cochlear capsule, there is no doubt that experimentally one can produce this large amount of new bone in the internal meatus by vitamin A depletion as pointed out in the paper. However, there is little clinical significance in such an observation.

COLLEGE OF PHYSICIANS OF PHILADELPHIA, SECTION ON OTOLARYNGOLOGY, AND THE PHILADELPHIA LARYNGOLOGICAL SOCIETY

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Regular Meeting, Nov. 17, 1943

Percussion Note of the Maxillary Sinus. Presented by DR. WILLIAM HEWSON.

This article was published in full in the ARCHIVES OF OTOLARYNGOLOGY (**38**:350 [Oct.] 1943).

DISCUSSION

DR. WALTER HUGHSON: It is a privilege to be asked to discuss this scholarly paper presented by my symphoniously named colleague. Dr. Hewson in his introductory paragraph draws attention to one of the fundamental lacks of modern specialized medical practice, namely, the reliance of the practitioner on newly developed diagnostic procedures and the abandonment of those technics which formerly were the true background of the "art of medicine." I see no prospect of the younger generation of specialists acquiring a broader background of clinical medicine. Indeed, with the present accelerated methods of teaching it is difficult to see how they will acquire any background at all except

from such a presentation as the one to which we have just listened. I agree most heartily with this plea for reemphasis of the truly clinical methods of physical diagnosis.

In making this statement I realize that my own position in the field of otology may well expose me to the criticism of being one of the worst offenders against this very thing. However, as Dr. Hewson points out, sound and its production and recognition belong primarily to the science of physics. Therefore one must supplement the clinical approach by every physical means available in the effort to arrive at exactness in diagnosis.

In his discussion of the "percussion note of the maxillary sinus" and the information which may be derived therefrom, Dr. Hewson has no intention, I feel quite sure, of advocating the abandonment of such routine procedures as transillumination, roentgen examination or any other procedure which might present, but urges the otolaryngologist to use the routine procedure of percussion, which, as he says, is "at hand" or, one might say more properly, "at one's finger tips."

I have seen Dr. Hewson demonstrate his technic, and certainly the proof of its efficacy is in the actual hearing of it. Happy would be the person in this particular climate from whose maxillary antrums Dr. Hewson could elicit a truly tympanitic note. A careful study of all the gradations of sound produced by percussion in relation to such factors as the actual size of the sinus, the actual density of the skull, the bony structures specifically involved, the type and the extent of the pathologic change present and the changes in note depending on development or subsidence of disease will add greatly to the otolaryngologist's skill in handling this difficult and still rather unsatisfactory situation.

From a pure tympanitic note one would find all degrees of change to a boardlike flatness. As Dr. Hewson says, external changes even of such slight nature as a temporary closure of the ostium may affect the clarity of the note. The entire subject is most interesting and intriguing.

This paper should revive interest in one's pleximeter and percussor fingers which, like the legs in the automobile era, have undergone serious loss of function.

Dr. William Hewson should be congratulated on his presentation.

DR. DOUGLAS MACFARLAN: Dr. Hewson's paper is unique and stimulating. It recalls to mind some interesting phonendoscopic work done on the skull in the past, which was reported by Dr. Nussbaum before this society many years ago.

Dr. Batson and I attempted to follow up the study but found that the resonance of the skull was highly variable. We checked the resonance to the frequency range by auscultation at various skull locations. The head was put in resonance by an activated bone receiver. A chronic thickening of the mucosa, as well as fluid in the cavities, throws the sound to one side. Of course, the bony sclerosis from chronic sinus infection will do the same.

I suggest, as an experiment in the percussion of the sinus, that the examiner use binaural auscultation tubes from the patient's ears to his own. Through these tubes the percussion note will be well transmitted, and outside disturbing noise will not interfere with the examiner's hearing. I have been using and recommending this method of auscultation for patency of the eustachian tube for some years.

Dr. Hewson is absolutely correct in his statement that no one diagnostic method is conclusive in cases of sinusitis. One should use and correlate them all.

DR. WILLIAM HEWSON: I want to thank the discussers for their remarks. I have listened to the ma illa. It does seem to me that percussion is such an e thing to use. Every one should try it.

The Absorptive Capacity of the Nose. Presented by DR. MAURICE SALTZMAN.

This article will be published in full in a later issue of the ARCHIVES.

DISCUSSION

DR. MATTHEW S. ERSNER: It is a privilege to discuss Dr. Saltzman's thesis. It is predicated on the normal physiologic function of the mucous membrane of the nose and the sinuses. He emphasized the importance of the goblet cells, the mucin, the mucous sheet, the ciliated epithelium and the lysozymes. Because of these elements, there is a paucity of absorption.

Infection, drugs and exposure to heat or to cold retard ciliated motility, and it is then that the surface of the nasal mucous membrane is vulnerable. The nasal mucous membrane has inherent qualities to absorb exotoxins or endotoxins, and it may also absorb medicaments when applied by spray, drops or tamponage.

It is interesting to note that the pharmacologists speak of many vasoconstrictor drugs for topical application to the nasal mucous membrane. However, they do not have any specific vasodilator drugs which can be applied directly to the nasal mucous membrane. The only drugs the physicians have are irritants, which produce congestion rather than dilatation. All are acquainted with the vasoconstrictor drugs, their local effects and occasionally the general toxic manifestations that result from their use. Occasionally one reads of intoxication due to cocaine, of sensitivity to ephedrine or toxic symptoms caused by epinephrine when the drugs have been absorbed through the nasal mucous membrane.

The newer nasal medicaments, such as the sulfonamide compounds, are incorporated with vasoconstrictor drugs such as ephedrine, neo-synephrine or other astringents. A great many patients will absorb the sulfonamide compounds when therapy is prolonged, and toxic symptoms are apt to be manifested.

In the light of present knowledge it seems best to proceed cautiously in the administration of a sulfonamide drug, particularly if therapy is prolonged. Therefore, hemograms should be taken so as to ascertain whether or not there is anemia or agranulocytosis. The Marshall test should also be performed frequently when such nasal medication is employed.

In my practice I have encountered 2 cases of blood dyscrasia due to the use of sodium sulfathiazole as a powder. I am convinced that the sinonasal mucous membrane not only responds to vasoconstrictors but also has inherent properties for absorption.

I should like to cite 2 other cases: Both patients had cancer of the epipharynx. Biopsies were made, and in order to prevent bleeding, postnasal packs were inserted into the epipharynx without packing the anterior nares. All blood clots were removed by aspiration, leaving the nose clean. In one case sulfanilamide crystals were instilled every four hours; in the other microcrystals were insufflated with the powder blower. Within six hours blood was collected from both patients for the Marshall test. In the first case 3.5 mg. of sulfathiazole per hundred cubic centimeters of blood was found; in the second case, 4.8 mg. Within twenty-four hours the

entration in the second case was 13 mg. per hun- cubic centimeters. Since the posterior nares were pletely blocked by the postnasal pack, the possibility the drug was swallowed and absorbed by the rointestinal tract is eliminated. The only avenue bsorption here was through the nasal mucous brane.

R. THOMAS F. FURLONG JR.: I should like to com- ent Dr. Saltzman on his excellent and interesting entation. I should also like to ask him the following tions: 1. For atrophic rhinitis, do you spray or ally apply estrogenic substances to the nasal mucous brane and what preparation do you use? 2. Do use an infra-red ray lamp to increase absorption should the use of the lamp precede or follow the ying or the topical application of the drugs?

R. SALTZMAN: I wish to thank the discussers. Dr. er mentioned the danger from absorption of the namide compounds which are being used extensively asal treatment. It appears that suspensions of high osity should not be used for instillation into the rums through the irrigating cannula, nor should preparations be employed in the displacement tment. O'Donnel's reported cases of toxicity are nite examples.

r. Furlong mentioned atrophic rhinitis and infra- ray therapy. My associates and I have had a derate amount of experience with amniotin (a prep- ion of noncrystalline estrogenic substances from the e of pregnant mares). One method of employing a-red ray therapy is as follows: Pledgets of cotton rated with 0.5 per cent ephedrine in isotonic solution odium chloride are inserted into the middle meatus between the inferior turbinate and the septum. h the medication in the nose an exposure to the p is given for about twenty minutes. We never erved any toxic absorption from this procedure, and results from the treatment were uniformly gratifying.

Series of Round Table Conferences on Rhinoplasty. First Conference: Anatomic Considerations. Presented by DR. KELVIN A. KASPER.

ny book on anatomy presents a picture of the more less perfect nose, which is, however, seldom found nature. Actually a hundred noses present a hundred iations from the normal.

n this ideal anatomy the nose is a triangular pyramid, row at the root, which lies between the orbits, and adest at the base, which at the surface is continuous h the upper lip.

The constituents of the nasal framework in descending er are the nasal bones, the bony and cartilaginous tum and the upper and lower lateral cartilages.

The root is formed by the articulation of the nasal es and the frontal processes of the maxillary bones h the frontal bone.

The nasal bones are in contact beneath with the bony cartilaginous elements of the septum and with the tilaginous septum and upper lateral cartilages at ir distal ends. Determining, as they do, the height the contour of the nose in the region of the orbit, it the nasal bones that largely control the nasal form. y set the line of the dorsum. They are convex ve and concave below.

Continuing from the perpendicular plate of the eth- id bone is the cartilaginous septum. This rests on floor of the nose, where its base occupies a groove the vomerine cartilage, immediately above the vomer. e body of the cartilage terminates in a straight line connecting the upper lip with the nose tip, thus accounting for an angle with the line of the dorsum.

From the cartilaginous septum where it emerges below the nasal bones, wings radiate outward toward the maxillas, providing support for the tissues of the nose, which widens as it descends from the region of the nasal bones. These wings are the upper lateral cartilages.

Below them and giving form to the nostrils and the tip, at which the nose ends, are the lower lateral cartilages. In their outward extension the lower lateral cartilages give form to the nostrils. Forming an angle at the level of the upper edge of the septum and slightly in front of the normal limit of its extension, they curve downward. It is to this curving that the nasal tip conforms.

Descending in the direction of the lip, this medial crus invests the tissues of the columella, whose soft tissues provide surface covering for the caudal end of the cartilaginous septum from the lip to the tip of the nose.

Over this framework, the skin and other covering tissues of the nose extend laterally from the dorsum, becoming confluent with the surface and tissues of the face. The skin of the nose varies in density. Over the nasal root it is thin and loose. At the nasal tip and the alae it is thick and adherent and is bound with thick connective tissue. Within the nasal orifices the covering is a mucous membrane, thick, highly vascular and firmly bound to the periosteum and perichondrium which it overlies. Because of this intimate adherence, fractures of the nose are almost invariably compound.

Regarding the muscles of the nose: Those extending from above elevate and dilate, those from below depress and contract, the nostrils. These are all supplied by branches from the facial nerve. The procerus muscle, which is continuous with the occipitofrontalis muscle, draws down the inner angles of the eyebrows and produces transverse wrinkles. It blends with the caput angulare. The caput angulare extends down the side of the nose from the inner margin of the orbit to the ala, by one division, and by another division to the lip. It acts to draw up the lip and dilate the nostril.

That principal agent in expressing contempt, the pars transversa of the nasalis muscle, arises from the maxilla above the incisive fossa. Fibers go upward and inward. On the bridge it is continuous with the procerus muscle and its fellow of the opposite side. It depresses the cartilaginous part of the bridge and compresses the alae. The dilator naris posterior and the dilator naris anterior are small muscles whose function is to enlarge the aperture of the nostril.

The compressor narium minor is a small muscle attached by one end to the cartilage and by the other to the integument at the end of the nose. The pars alaris of the nasalis muscle (depressor alae nasi) arises over the incisive fossa and the fibers ascend to be inserted in the septum and the back part of the ala. Its action is to draw the ala downward, contricting the aperture of the nostril.

DISCUSSION

DR. BENJAMIN H. SHUSTER: It was my suggestion to the members of the executive committee, which they kindly accepted, that there should be at these meetings round table conferences on the subject of rhinoplasty. There appears to be a number of otolaryngologists who have become interested in this subject. At the last meeting of the Academy of Ophthalmology and Otolaryngology, an exhibit by Drs. R. A. Luongo and

Fomon on rhinoplasty received the first prize, indicating the amount of interest in the subject.

A word about the attitude of physicians on rhinoplasty and that of rhinoplastic surgeons. Until recently rhinoplasty has been looked on by many as in the domain of quackery. I believe that this attitude is gradually being dispelled as more ethical men become interested in rhinoplasty and begin to treat it like any other ethical procedure, in an ethical way. As for the attitude of rhinoplastic surgeons, in many instances it could not exactly be called enthusiastic. There appears to be an aroma of secrecy about the entire subject. One interested in learning the work often finds difficulty in being permitted to observe those surgeons doing their work. Presumably it is to keep others from learning and becoming competitors. This attitude, too, I believe, is gradually diminishing and I hope it will disappear completely soon. Dr. Fomon, who is training a number of otolaryngologists in rhinoplasty, has contributed much toward dispelling the mystery. Those of the members who are interested in this work could also help by interchanging ideas as is expected at these conferences, and in this way help each other.

I classify patients desiring plastic surgery into three groups: First are those who have deformities due to trauma, and such deformities are frequent in this day and age. They certainly ought to get the benefit of this type of work. It is the rhinologist's duty to perfect himself in rhinoplasty so that he may offer the best service. Second are the patients whose noses are congenitally different from the average noses and who feel self conscious about it. This group certainly are entitled to scientific consideration. Third, there is a group of patients who have little deformity but wish to make a change from one minor defect or another. There is no objection to treating this third group, but when one does one may expect a "headache." Most of these people are emotionally unstable, never know what they want and will not be satisfied with what they get.

If rhinologists decide to do this work, I believe, it ought to be presented to the patient for what it is: correction of a deformity. One should not resort to subterfuge excuses such as the importance of the change with respect to respiratory function or to the patient's ego and thereby to his career. There are few noses whose respiratory function cannot be corrected without resorting to external surgical alterations, and to insist that a patient go through a surgical operation on the basis of improving the function of his nose to my mind is in many instances on the shady side. As far as a career is concerned, if a man's career depends on the shape of his nose, I doubt, if that career is w much and repairing his nose will not help him.

Turning to the subject of anatomy, it is impor that this subject be mastered from the standpoi the parts to be repaired. If one could see the nose without the cutaneous covering, it would not an expert to change the parts to any shape de One might remove or add parts as one saw fit wi having much imagination. As plastic operations all don subcutaneously, imaginative visualizatio the part becomes imperative. There are three sions of the nose as far as rhinoplasty is conce and they are:

1. The nasal bones and the adjoining ascending p esses of the superior maxillas. These often have t manipulated to narrow the nose, and defects are countered in areas near the glabella which, if realized, leave the bridge broadened at the top. L bits of bony tissue have to be removed to overc impediments to narrowing the bridge.

2. The upper lateral cartilages. These may be vated, depressed or trimmed as seems necessary produce the desired shape, but one must definitely in mind where the fault with these cartilages lies (i the skin), so that the proper area may be manipul

3. The last part to be considered is the one contain the lower lateral cartilages. Improper manipula of these cartilages results in a distorted nasal tip; proper manipulation almost any desired shape ca produced.

The septum, too, in its relation to the cartilages nasal bones must be considered, as it may prot or sag and requires proper attention. If not visual properly, tissue may be removed from the lateral n wall instead of the septum, with consequent distor and a poor result.

These remarks are sufficient to indicate the imp tance of visualizing the anatomy of the structures neath the skin.

Dr. Kasper's paper, therefore, deserves much atten and study, particularly in relation to this most import subject of the anatomic relationships.

DR. THOMAS F. FURLONG JR.: Will plastic surg come to the forefront after the war?

MAJOR STEWART: Yes. The present policy in Army is not to emphasize cosmetic surgery except cases of marked disfigurement. The rhinoplastic s geons are spending most of their time correcting in tional deformities, aiding the soldiers back to useful There will be a great deal of plastic surgery after war and it will be done in the United States Vete Administration Facilities.

Book Reviews

l Pathology. Kurt H. Thoma, D.M.D., professor f oral surgery and Brackett professor of oral athology, Harvard University; oral surgeon and hief of dental service, Massachusetts General Hosital; oral surgeon, Brooks Hospital; dental surgeon, oston Dispensary Dental Department; consultant n oral surgery, Boston Dispensary Tumor Departent and Joseph H. Pratt Diagnostic Hospital; conulting oral surgeon, New England Baptist Hospital, nd consulting oral surgeon, Beth Israel Hospital. dition 2, revised. Pages 1,328, with 1,388 illustraons, including 128 in color. Price $15. St. Louis: . V. Mosby Company, 1944.

homa in this book deals with the teeth, the jaws the oral mucosa from the point of view of pathology. esion within the oral cavity may be due to local es, such as infection or a neoplastic process; or it be related to neighboring fields, such as the nose, pharynx, the larynx or the ear; or it may be an ression of a somatic disease, which may be nutrial, metabolic, hemopoietic or of virus origin.

he book is divided into twelve parts. The first three concerned with hereditary, endocrine and nutritional ences on the development of the teeth, the jaws the face and the modifications of such growth and lopment by local factors and systemic disease.

Parts 4, 5 and 6 present the pathology of the teeth. Part 7 concerns periodontal disease, covering infections of the gums and of the supporting structures of the teeth. Part 8 is of particular direct interest to the otolaryngologist, as it is a study of the interrelationships of dental disease and disease of neighboring and distant parts of the body. Part 9 covers traumatic and infectious diseases of the jaws, systemic diseases with manifestations in the jaws and abnormalities of the temporomandibular joint. Part 10 consists of four chapters on tumors of the jaws and the oral mucosa. Part 11 is entitled "Diseases of the Oral Mucosa, Lips and Tongue." Many systemic diseases have signs and symptoms in the oral cavity. The colored photographs in this section are valuable.

Part 12 deals with the inflammatory diseases and tumors of the salivary glands and mucous glands.

This book was written by an authority whose original research, erudition and experience preeminently qualify him to undertake such a work. Technically, the paper is good, the printing clear, the arrangement of material excellent and the matter easily accessible, with a bibliography at the end of each chapter and valuable additions of many illustrations in black and white and color. It should be of great interest to the internist and to any one dealing with the head and the neck.

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NATIONAL

American Medical Association, Scientific Assembly, Section on Laryngology, Otology and Rhinology

Chairman: Dr. Claude C. Cody, 1304 Walker Ave., Houston, Texas.

Secretary: Dr. Louis H. Clerf, 1530 Locust St., Philadelphia.

Place: Chicago. Time: June 12-16, 1944.

American Academy of Ophthalmology and Otolaryngology

President: Dr. Lawrence T. Post, 508 N. Grand Blvd., St. Louis, Mo.

President-Elect: Dr. Gordon B. New, Mayo Clinic, Rochester, Minn.

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American Broncho-Esophagological Association

President: Dr. Robert L. Moorhead, 125 Ramsen St., Brooklyn.

Secretary: Dr. Paul Holinger, 700 N. Michigan Ave., Chicago.

Place: New York. Time: June 6, 1944.

American Laryngological Association

President: Dr. Charles J. Imperatori, 108 E. 38th, New York.

Secretary: Dr. Arthur W. Proetz, Beaumont Bl[d], St. Louis.

Place: New York. Time: June 7-8, 1944.

American Laryngological, Rhinological and Otological Society, Inc.

President: Dr. H. Marshall Taylor, 111 W. Adams, Jacksonville, Fla.

Secretary: Dr. C. Stewart Nash, 708 Medical Arts Bldg., Rochester, N. Y.

Place: New York. Time: June 9-10, 1944.

SECTIONS:

Eastern.—Vice President: Dr. Westley M. Hunt, 9[0] 5th Ave., New York 21.

Southern.—Vice President: Dr. William C. Warren, 478 Peachtree St. N. E., Atlanta, Ga.

Middle.—Vice President: Dr. Fred W. Dixon, 20[0] 9th St., Cleveland.

Western.—Vice President: Dr. Simon Jesberg, 5[0] Lucas Ave., Los Angeles.

American Otological Society

President: Dr. Wesley C. Bowers, 17 E. 61st, New York 21.

Secretary: Dr. Isidore Friesner, 101 E. 73d St., [N]ew York.

Place: New York. Time: June 5-6, 1944.

* Secretaries of societies are requested to furnish the information necessary to keep this list up to date.

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